

Chapter 16

Dermatologic Diseases

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I. DIAGNOSTIC APPROACH TO DERMATOLOGIC DISEASE

A. History and clinical findings. Because an accurate history is often difficult to obtain, use of a detailed questionnaire is helpful. Important points to consider when obtaining the history include:

1. Age, breed, sex, and color of the animal
2. Duration, characteristics, and changes in the primary complaint
3. Presence or absence of pruritus
4. Seasonality of the condition
5. Type of housing
6. Other affected animals
7. Internal and external parasite control program
8. Systemic or topical medication used for skin conditions or recent problems
9. Other medical problems

B **Physical examination.** The overall condition of the animal should be assessed.

1. Lesions. The distribution (e.g., generalized, localized, specific body regions) and type (e.g., primary, secondary) of skin lesions should be noted.
 - a. Primary lesions include papules, macules, nodules, tumors, pustules, wheals, and vesicles.
 - b. Secondary lesions include scales, crusts, excoriations, erosions, ulcers, lichenification, pigmentation changes, and fissures.
2. The skin surface should be palpated to determine features not easily visible (e.g., crusts beneath hair, dryness, ability to epilate hairs, presence of peripheral lymphadenopathy).
3. The mucous membranes should be examined.

C. **Differential diagnoses.** The skin can react to a variety of insults in limited ways. This may make reaching a diagnosis difficult. Lists of differential diagnoses are based on information gained from the history and physical examination. Several causes of the presenting complaint are usually considered, and the diagnosis is confirmed by either testing the hypothesis or measuring any response to a specific therapy.

D **Diagnostic techniques**

1. Skin scraping is often used to demonstrate the presence of external parasites (e.g., mites, lice).
 - a. Materials for skin scraping include a sterile container, mineral oil, a #10 scalpel blade, glass slides, and coverslips.
- b. **Procedure**
 - (1) Areas with hair (e.g., fetlock) should be lightly clipped before scraping.
 - (2) Multiple superficial scrapings covering a large surface area should be obtained. Skin scrapings should be deep enough to induce capillary oozing to recover deep skin-dwelling mites.
 - (3) The collected material is placed in a container until a microscopic examination can be performed. The sample is then placed on a glass slide and finely dispersed in mineral oil to provide a confluent layer without air bubbles.

2. Wood's lamp examination is not useful in large animals. *Microsporum canis* is the only dermatophyte that fluoresces and then only 50%–60% of the time. This organism usually affects only dogs and cats. Horses and cattle are most commonly infected with *Trichophyton* species, which do not fluoresce. Therefore, a negative Wood's lamp examination does not rule out dermatophytosis.
3. **Dermatophyte culture.** Animals with focal or generalized alopecia with or without scaling or crusting are candidates for a dermatophyte culture.
 - a. Materials for the test include a dermatophyte test medium, 70% isopropyl alcohol, mosquito forceps, a #10 scalpel blade, and sterile empty containers, such as evacuated blood collection tubes.
 - b. Procedure
 - (1) Collecting the sample. The forceps and each lesion to be sampled should be wiped gently with isopropyl alcohol and allowed to dry in order to remove as many bacterial and saprophytic contaminants as possible. Multiple, small, scaling and lightly crusted lesions should be sampled. Broken hairs, scales, and crusts from the periphery of the lesion are collected. A blade may be useful in scraping scales and debris from the skin surface. The forceps are used to pluck and sample broken hairs. Samples should be stored in separate sterile containers.
 - (2) Testing the sample. Samples should be removed from containers with sterile forceps in a clean working area and gently pressed onto, but not buried beneath, the culture medium. The top of the culture dish should be loosely replaced to allow sufficient ventilation. Most dermatophytes grow at room temperature with the exception of *Trichophyton verrucosum*, which requires incubation at 37°C.
 - (3) Results. The dermatophyte test medium is an amber-colored Sabouraud's dextrose agar, containing phenol red, a pH indicator, as well as several antibacterial and antifungal agents to inhibit the growth of contaminant organisms. Dermatophytes preferentially use the protein in the medium as they begin to grow, producing alkaline metabolites that cause the medium to turn red. The dermatophyte colony is typically a white-to-beige, powdery-to-fluffy growth. The red color change on Sabouraud's and colony growth characteristics should occur concurrently for positive identification.
4. Potassium hydroxide (KOH) preparations may permit the immediate diagnosis of a dermatophytosis. However, negative results do not rule out dermatophytosis. Examination of KOH preparations requires considerable expertise because fungal elements may be overlooked, and numerous artifacts (e.g., fibers, cholesterol crystals, oil droplets) may be confused with fungal elements. A dermatophyte culture should always be performed in conjunction with a KOH preparation.
 - a. Materials for the test include mosquito forceps, a #10 scalpel blade, a sterile empty container, glass microscope slides, coverslips, a Bunsen burner, and the clearing solution.
 - b. Procedure. Hairs and scales should be collected from the periphery of several lesions using the mosquito forceps and the blade. A drop of clearing solution is placed on a glass slide, hairs and scales are added to the solution, and a coverslip is placed over the material. The purpose of the clearing solution is to dissolve hard keratin and bleach the melanin of the hair shaft so that hyphae and arthroconidia can be identified more readily. Clearing solutions include 15% KOH (heated for 15–20 seconds or allowed to stand at room temperature for 30 minutes) or a KOH and dimethyl sulfoxide (DMSO) solution, which permits immediate examination. The slide is scanned with the 10-times (10x) objective in search of abnormal hairs with a fuzzy internal structure. High-power examination of these hairs may demonstrate hyphae, which usually have uniform width and septae. Bead-like chains of arthroconidia may be seen as well.
5. Acetate tape preparations are used to diagnose *Oxyuris equi* or *Chorioptes equi* infestation.
 - a. Materials used include acetate (nonfrosted) tape, mineral oil, and glass microscope slides.
 - b. Procedure. A piece of tape is pressed onto several areas in the anal and perianal region and then removed and placed (adhesive side down) on a glass slide liberally coated with mineral oil (which will clear debris and facilitate the examination of the preparation).
6. **Dermatophilus preparation.** Crusted lesions accompanied by matting of the coat in horses and ruminants are clinical findings for dermatophilosis. Because suppuration underlies the crust, direct smears of the exudate must be made, stained, and examined on a glass slide for characteristic bacteria. Crusts can be removed from the patient, macerated with scissors, and mixed with several drops of water on a glass slide. After crusts have been softened in water, they should be crushed with the top of an applicator stick. Excess debris should be removed, the slide should be air dried, heat fixed, and then stained with Gram's, Giemsa, or Wright's stain.
7. Biopsy for routine histopathology. The types of lesions to be biopsied include suspect neoplastic lesions, persistent ulcers, dermatoses not responding to appropriate therapy, and any unusual or serious lesions. Fully developed primary lesions should be selected. Biopsy for histopathology is usually unrewarding for chronic (e.g., lichenified, hyperpigmented) lesions. If possible, multiple lesions should be biopsied.
 - a. Materials for performing a skin biopsy include 6-mm and 4-mm biopsy punches, a #15 scalpel blade, sharp scissors, Adson forceps, needle holders, 2-0–3-0 nonabsorbable suture material, 2% lidocaine, a 3-ml syringe with a 22- to 25-gauge needle, a tongue depressor, gauze, and 10% buffered formalin.
 - b. Procedure
 - (1) Preparation. Surgical preparation (i.e., shaving and scrubbing) will remove crusts and epithelial tissue that are important for diagnosis and should not be performed. Local anesthesia is accomplished by inserting the needle at the margin of the lesion and infiltrating lidocaine subcutaneously.
 - (2) Biopsy techniques. Excisional (either punch or elliptical) skin biopsy techniques can be used.
 - (a) Punch biopsies are used to assess a single nodule. Most lesions can be sampled with a 6-mm disposable biopsy punch. When a biopsy has been obtained, the site can be sutured with simple interrupted sutures or a cruciate stitch. When obtaining biopsies of ulcerations, the biopsy should include normal tissue, the leading edge of the lesion, and abnormal tissue.
 - (b) Elliptical biopsies. Punch biopsies are not appropriate for vesicular, bullous, or ulcerous lesions. Instead, an elliptical biopsy is preferred. Elliptical biopsies, which are obtained with a scalpel, should be mounted with gentle pressure on a small piece of tongue depressor or cardboard to prevent curling.
8. Biopsy for immunofluorescence testing. Immunofluorescence testing is used as an adjunct to conventional histology in patients with suspected immune-mediated skin disease (e.g., pemphigus foliaceus, bullous pemphigoid, discoid lupus erythematosus, systemic lupus erythematosus, some forms of vasculitis). The technique is similar to that used to obtain biopsies for histopathology, but other fixatives are used. The diagnosis of immune-mediated skin disease is made on the basis of both histologic and immunofluorescent findings.
9. Onchocerca preparation. Horses with lesions of alopecia and scaling (with or without pruritis) that include diffuse ventral midline dermatitis and/or facial, cervical, thoracic, or proximal forelimb lesions are candidates for *Onchocerca* infestation. Many horses will have microfilariae present in the dermal tissue without any evidence of skin disease. A positive preparation only suggests that *Onchocerca* organisms are the cause of the dermatitis.

- a. Sample. If possible, a lesion should be sampled in a region where microfilariae are not recovered in high numbers in normal horses (e.g., avoid the ventral midline). The sample should be obtained with a 10-mm punch biopsy and split in half. Half of the sample should be put in formalin, and the other half should be placed on a dampened gauze sponge in a tightly closed container until a preparation can be made.
 - b. Preparation. A small piece of tissue that includes dermis is placed on a glass slide, minced with a scalpel, and a few drops of nonbacteriostatic saline are added. The slide is then incubated at room temperature for 15 minutes.
 - c. Results. The slide is scanned with a microscope (4x objective) along the margins of tissue debris, searching for indications of motion in the saline. If the characteristic "whiplash" movement of the parasite is noted, a higher power objective should be used. Microfilariae are slender and delicate, measuring approximately $8\ \mu\text{m} \times 220\ \mu\text{m}$.
10. Bacterial culture. A bacterial culture should be requested when folliculitis or bacterial pyoderma are suspected. Normal skin is heavily contaminated by bacteria; therefore, cultures must be carefully taken and interpreted. Cultures of open, draining lesions generate misleading bacteriologic data.
- a. Samples
 - (1) Intact samples. Intact nodules, pustules, and abscesses may be aspirated with a needle and syringe or punctured with a scalpel and swabbed with a culturette after gently cleaning the overlying skin with alcohol and allowing it to air dry.
 - (2) Biopsy samples. When intact lesions containing pus are not available for sampling, a culture of surgical biopsy specimens is preferred. Papules, plaques, nodules, and areas of diffuse swelling may be surgically prepared and punch or excision biopsies taken with aseptic technique.
 - b. Preparation. Samples for culture are routinely plated on blood agar and inoculated in thioglycolate broth to encourage the growth of any anaerobes.

II. ENVIRONMENTAL AND MECHANICAL DERMATOSES

A. Frostbite is a skin injury resulting from exposure to excessive cold.

1. Patient profile and history
 - a. Neonates and sick, debilitated, or dehydrated animals are most prone to frostbite injury.
 - b. Lesions are seen on the extremities after cold weather.
2. Clinical findings include erythema, scaling, and alopecia affecting the ears, tails, teats, scrotum, and feet. Severe cases are characterized by necrosis, dry gangrene, and sloughing. Affected skin usually lacks feeling.
3. Pathogenesis. Frostbite is more common in animals with poor circulation (e.g., as seen with ergotism, fescue toxicosis, vasculitis) or in animals that are unable to seek shelter.
4. Diagnostic plan. History and clinical signs determine the diagnosis.
5. Therapeutic plan
 - a. Mild cases. Treatment is unnecessary for mild cases.
 - b. Severe cases. Rapid thawing in warm water (41°C – 44°C) followed by the application of bland, protective ointments and creams is the treatment of choice for severe cases. Where there has been necrosis and sloughing of skin, topical wet soaks and systemic antibiotics should be used. Surgical excision should be postponed until an obvious boundary is present between viable and nonviable tissue.

6. Prevention. After it has been frozen, tissue may have an increased susceptibility to cold injury.

B. Thermal injuries (burns). Occasionally, burns are seen in large animals in association with barn fires, brush fires, electrocution, or lightning strike. The prognosis is poor for animals with burns covering more than 50% of the body surface. Treatment includes:

1. Cleansing with mild soap and water 2–3 times daily
2. Initial surgical debridement
3. Daily hydrotherapy
4. Topical antibiotics
5. Systemic nonsteroidal anti-inflammatory agents

C. Primary irritant contact dermatitis

1. Patient profile and history. The animal may present with dermatitis of the face, distal extremities, or ventrum.
2. Clinical finding. This dermatitis ranges in severity from erythema to papules, edema, scaling, vesicles, erosions, ulcers, necrosis, and crusts. Pruritus and pain are variable. Self-trauma resulting from the irritation can cause alopecia, lichenification, and scarring. Leukotrichia and leukoderma may be permanent sequelae.
3. Etiology. Primary irritants invariably cause dermatitis if they come into contact with skin in sufficient concentration for a long enough time. No sensitization is required. For example, leukoderma around the mouth of horses is commonly caused by the rubber bit.
 - a. Caustic substances may be either acids or alkalis. Crude oil, diesel fuel, turpentine, leather preservatives, mercurials, blisters, and wood preservatives are all primary irritants.
 - b. Moisture is an important predisposing factor because it decreases effectiveness of normal skin barriers. Causes for excessive moisture include body excretions (i.e., feces, urine), a damp environment, and wound secretions. Conditions that cause excessive moisture include:
 - (1) Scalding of the perineum in diarrheic calves or foals
 - (2) Flank or udder fold dermatitis in lactating dairy cows
 - (3) Ovine fleece rot
 - c. Other causes of primary contact dermatitis include improperly used topical parasiticides and irritating plants (e.g., sneezeweed, stinging nettle).
4. Diagnostic plan. The nature of any contact dermatitis can usually be inferred from the distribution of the lesions.
 - a. Muzzle or extremities—plants and environmental substances (e.g., fertilizers)
 - b. Single limb—irritating medications (e.g., blisters)
 - c. Face and dorsum—sprays, dips, and wipes
 - d. Perineum and rear legs—urine and feces
 - e. Tack-associated areas—preservatives, dyes, and polishes
 - f. Ventrum—bedding and filthy environment
5. Laboratory tests. A skin biopsy reveals varying degrees of superficial dermatitis (e.g., hyperplastic, spongiotic), with neutrophils or mononuclear cells predominating.
6. Therapeutic plan. Irritants should be identified and eliminated. Any residue should be removed with large amounts of water and gentle cleansing soaps. Astringent soaks (e.g., aluminum acetate, magnesium sulfate in water) are applied to moist, oozing lesions for 5–10 minutes three times daily. Topical antiseptics, antibiotics, and/or glucocorticoids may be considered in cases of severe or significant inflammation.
7. Prognosis. Removal of the irritant results in remarkable improvement of the dermatitis in 7–10 days.

D. Chemical toxicosis. Ingestion or exposure to some chemicals or agents produces skin disease.

1. Mercury poisoning produces acute gastroenteritis and death if ingested in sufficient quantities. Mercury also causes alopecia with the loss of long hairs in the tail and mane if ingested in smaller quantities over a longer time.
2. Iodine (or iodides) causes epiphora, nasal discharge, lacrimation, and seborrhea sicca with partial alopecia over the dorsum, head, neck, and shoulders. There is usually a history of sodium iodide or potassium iodide administration.

E. Photodermatitis

1. Sunburn
 - a. Patient profile and history. Sunburn produces a dermatitis of lightly pigmented skin (e.g., Saanen goats, white pigs).
 - b. Clinical findings. Include erythema and scaling. Severe burning causes exudation, necrosis, and crust formation. Sunburn is common on the lateral aspects of the udder and teats of white goats that are turned out in summer and along the back and ears of white-skinned pigs.
 - c. Laboratory tests. A skin biopsy reveals superficial perivascular dermatitis, dyskeratotic keratinocytes in superficial dermis, and basophilic degeneration of elastin.
 - d. Prevention. As in all species, initial exposure to sunlight should be limited. Sunscreen may be applied to susceptible animals if warranted.
2. Photosensitization is characterized by acute onset of erythema, edema, and variable degrees of pruritus and pain.
 - a. Clinical findings. Cutaneous lesions are often restricted to unpigmented, sparsely-haired areas but may extend to dark-skinned areas in severe cases. The eyelids, lips, face, ears, perineum, and coronary band region are commonly involved. Vesicles and bullae develop that progress to oozing, necrosis, skin sloughing, and ulceration.
 - b. Pathogenesis
 - (1) There are three basic features for all types of photosensitization:
 - (a) Presence of a photodynamic agent in the skin
 - (b) Concomitant exposure to a sufficient amount of certain wavelengths of ultraviolet light (UVL)
 - (c) Cutaneous absorption of UVL, which is facilitated by a lack of pigment in the haircoat
 - (2) Molecules of photodynamic agents absorb light energy at a specific wavelength and enter a higher energy state. In the presence of oxygen, excited molecules produce free radicals, which cause structural damage of cell and lysosomal membranes. This in turn causes the release of hydrolytic enzymes and other chemical mediators of inflammation.
 - (3) Classification of photosensitization
 - (a) Primary photosensitization. The photodynamic agent is preformed or produced metabolically in the body and may be acquired by ingestion, injection, or contact (Table 16-1).

TABLE 16-1. Primary Photosensitization in Large Animals

Photodynamic Plant Species	Chemicals
St. John's-wort	Phenothiazine
Buckwheat	Thiazides
Spring parsley	Acriflavines
Perennial rye grass	Rose bengal
Burr trefoil	Methylene blue
	Sulfonamides
	Tetracyclines

- (b) Hepatogenous photosensitization. Phylloerythrin, a porphyrin compound formed by the microbial degradation of chlorophyll in the gut, is normally conjugated in the liver and excreted in the bile. Liver dysfunction, biliary stasis, or both may cause an accumulation of phylloerythrin in the blood and body tissues with resultant photosensitization.
- (c) Photosensitization due to aberrant pigment synthesis (porphyria) is characterized by an accumulation of photodynamic porphyrins in the blood and body tissues as a result of aberrant pigment synthesis.
- (d) Photosensitization of unknown mechanism is associated with the ingestion of lush pastures (e.g., clover, alfalfa, vetch, oats).
- c. Diagnostic plan. Diagnosis is based on a thorough clinical examination and history, including information on the diet, pasture, and any drug therapy. The number of animals at risk should be compared with the number of animals affected. If many animals are affected, this may indicate a photosensitivity, whereas one affected animal may suggest photoallergy. Liver tests should be performed when the clinician suspects hepatogenous photosensitization (see Chapter 5 I B 1).
- d. Therapeutic plan. Therapy should be aimed at the primary cause. The source of the photodynamic agent should be identified and eliminated, sunlight should be avoided, and symptomatic therapy should be provided for hepatic disorders.
 - (1) Glucocorticoids or nonsteroidal anti-inflammatory drugs (NSAIDs) may be used to ameliorate signs.
 - (2) Systemic antibiotics may be necessary if there is secondary pyoderma.
 - (3) Surgical debridement may be employed for severe necrosis and skin sloughing.
- e. Prognosis. The prognosis is favorable for animals with primary photosensitization but is poor for those with hepatogenous photosensitization.

III. NUTRITIONAL DERMATOSES

A. Ergotism

1. Patient profile. This condition affects cattle, most commonly in the cooler months.
2. Clinical findings. The two clinical syndromes of ergotism include neurotoxicity and a form of chronic gangrene. With the gangrenous form, early edema of the coronary regions (most often the hindlegs) progresses to an abrupt line of ischemic necrosis (i.e., dry gangrene), usually in the mid-metatarsus area. The distal limb is cold and insensitive and eventually sloughs. Occasionally, ear tips and the tail tip are affected. Severe diarrhea often accompanies necrosis of the extremities.
3. Etiology and pathogenesis
 - a. *Claviceps purpurea* is a fungus that infests many cereal grains and crops under warm, moist growing conditions. These harvested grains are then used in the winter stabling period, and animals (mainly cattle) become affected 2–4 months after exposure.
 - b. Ergot alkaloids in fungus-contaminated feedstuffs cause vasoconstriction and capillary endothelial damage, with resultant ischemic necrosis and gangrene.
4. Diagnostic plan and laboratory tests. The presumptive diagnosis is based on clinical signs, history, environment, and finding blackened, fungus-contaminated feed. The diagnosis is confirmed by necropsy of affected animals and the analysis of suspected feedstuffs.

5. Differential diagnoses for the clinical signs include gangrene, frostbite, and trauma.
6. Therapeutic **plan and prevention**. Treatment is rarely attempted, and affected animals usually are culled. Ergot-contaminated grains or feedstuffs should not be fed to cattle. As a general rule, grain should not contain more than 0.1% ergot-contaminated heads if it is to be used for animal feed.

B. Fescue **toxicosis** (fescue **foot**) is virtually identical to poisoning by *Claviceps purpurea*, but fescue foot occurs specifically in cattle or sheep that are grazing fescue grass (tall fescue) pastures contaminated with *Acremonium coeniaphilum*.

C Zinc deficiency

1. Patient profile. Zinc deficiency is most often seen in cattle, sheep, and goats.
2. Clinical findings
 - a. Skin manifestations include a dull, rough haircoat and parakeratosis with scaling, crusting, and alopecia, particularly over the face, ears, eyes, distal limbs, and mucocutaneous junctions.
 - b. Generally, the animal has poor growth, stiff or swollen joints, and a decreased resistance to infections.
3. Etiology. A zinc-deficient diet or diets high in chelating minerals may cause this condition.
4. Diagnostic plan and laboratory tests
 - a. Response to therapy is the best diagnosis.
 - b. Serum and hair levels for zinc can be low or normal; thus, these tests are not diagnostic.
 - c. A skin biopsy shows evidence of parakeratotic hyperkeratosis.
5. **Therapeutic plan**. Modern diets are usually well balanced for trace minerals such as zinc.
 - a. Cattle should get 2–5 g zinc sulfate daily.
 - b. Sheep should get 40 mg zinc sulfate daily.

D. Copper deficiency

1. Patient profile. Copper deficiency affects mainly cattle and sheep.
2. Clinical findings
 - a. Affected animals have stunted **growth**, diarrhea, anemia, infertility, lameness, loss of wool crimp (in sheep), and heart failure.
 - b. Skin changes include a rough, brittle, faded haircoat (speckled in appearance), which the animal itches and licks.
 - c. Dark hairs may become light, particularly around the eyes, giving animals a "spectacled" appearance.
3. Etiology and pathogenesis. There is a dietary deficiency of copper or a secondary molybdenum excess. Copper is essential for the function of many oxidative enzymes, and evidence of dysfunction of these systems may be seen in the skin and haircoat (e.g., failure of pigmentation).
4. Diagnostic plan and laboratory tests
 - a. **Serum** copper levels should be less than 0.7 µg/ml to be diagnostic but might often be normal in affected animals.
 - b. **Liver** copper levels from a biopsy specimen seem to be more reliable and are evidence of herd copper status.
5. Therapeutic plan and prevention. Modern rations with attention to mineral supplementation should limit the likelihood of copper deficiencies.
 - a. Oral copper therapy. Copper sulfate is administered once weekly for 3–5 weeks at the following levels. Be aware that copper toxicity may occur at these levels.

- (1) Calves—4 g
 - (2) Cattle—6–10 g
 - (3) Sheep—1.5 g
- b. Alternative forms of therapy and prevention include injections and slow-release oral, copper bullets.

E. Selenium toxicosis

1. Clinical findings. Acute poisoning may result in sudden death, whereas chronic poisoning (alkali disease) produces hair loss and laminitis. Hair loss is most pronounced from the tail (in cattle and horses) and the mane (in horses).
2. Etiology. Selenium poisoning is caused by the ingestion of toxic levels of inorganic or organic selenium. Sources include supplemented feeds and plants that concentrate selenium from soils (so-called convertor or indicator plants, such as *Astragalus* species and *Haplopappus* species).

IV. VIRAL DERMATOSES

A. Introduction

1. Clinical findings. Many pox viruses that cause skin lesions begin as erythema and progress through papule, vesicle, pustule, and scab stages before resolving.
2. Diagnostic plan and laboratory tests. Cutaneous lesions may be the only manifestation of viral infection. Guidelines for obtaining samples for examination are as follows:
 - a. Samples should be stored at 4°C in a transport medium.
 - b. Samples should be taken from more than one lesion in more than one animal.
 - c. Areas for sample collection should be washed with water or saline but not with alcohol because it inactivates most viruses.
 - d. Specimen collection of the skin and mucous membranes should be extended to the periphery and base of the lesion.
 - e. Tissues should be submitted for both electron microscopy and virus isolation.

B. Specific conditions

1. Cowpox is a benign but contagious skin disease.
 - a. Clinical findings. This disease is characterized by typical pox-like lesions on the teats and udders of cows.
 - b. Etiology. The source of infection may be infected cows or milkers that were recently vaccinated against smallpox (vaccinia virus). Pain from the lesions may cause failure of milk letdown and mastitis.
 - c. Therapeutic plan. Treatment consists of the local application of soothing creams.
2. **Pseudocowpox** is similar to cowpox but is more common and cycles in the herd with a prolonged clinical presentation and healing time (up to 18 months). It is caused by a parapoxvirus and seen primarily in lactating heifers. The infection is **zoonotic** and causes milker's nodules in humans.
 - a. Clinical findings. Lesions are restricted to teats of lactating cows. Edema and erythema is followed by papules (within 48 hours), then a dark red scab that becomes raised by granulation tissue (the central area appears umbilicated). The center desquamates, leaving a raised ring of crust in the shape of a horse shoe. The granuloma may persist for months.
 - b. Diagnostic plan and laboratory tests. Diagnosis is based on the clinical findings and history and supported by electron microscopy examination of vesicle fluid (which shows intracytoplasmic inclusions) or virus isolation.

- c. Therapeutic plan and prevention. Treatment of affected cows has little effect, but the spread of disease is lessened by milking affected cows last, using teat clips, and reducing teat trauma.
3. Bovine ulcerative mammillitis is caused by bovine **herpesvirus** type 2, and the resulting lesion is ulcerative in nature.
 - a. Clinical findings. The teats of young, milking animals are most often affected with painful edema, vesiculation, and skin sloughing. There is no systemic involvement, but **mastitis** occurs because of the very painful nature of the lesions and subsequent failure of milk letdown.
 - b. Diagnostic plan and laboratory tests. The diagnosis is based on clinical signs and supported by virus isolation in a bovine tissue culture (either from vesicle aspirate or lesion biopsy). **Histopathology** of biopsy specimens shows intranuclear inclusion bodies in early lesions.
 - c. Therapeutic plan. Treatment is nonspecific and focuses on softening and protecting the lesions from secondary infections through the use of antibiotic creams.
4. **Sheeppox** and **goatpox** are contagious, pox-like dermatides that occur mostly outside of North America.
5. **Swinepox** is a common benign disease that affects the ventral abdomen of pigs. The **poxvirus** is transmitted by lice and affects mainly young pigs. Mortality is negligible, and treatment is not practiced.
6. **Ovine** viral ulcerative **dermatosis** is a venereal and skin disease caused by a **poxvirus**. This dermatosis is most common in the western United States during the breeding season. Lesions consist of granulating ulcers on the genitalia, lips, or legs. Treatment is nonspecific, and prevention focuses on hygiene management.
7. Equine viral papular dermatitis is reported sporadically and produces asymptomatic **papules** over the **body**. The disease is self-limiting but must be differentiated from other conditions, such as furunculosis.
8. **Horsepox** is rare and reported only in Europe.
9. Contagious **ecthyma** (viral pustular dermatitis, **sore** mouth, **orf**) is a **parapoxvirus** that affects sheep and goats, and this disease may be transmitted to humans. The organism remains infectious in the environment for extended periods (i.e., years); thus, when a farm has been contaminated, all incoming naive animals are at risk of disease. There is high morbidity on the first exposure of a flock. Immunity is strong and usually lifelong, but passive transfer is not effective in protecting lambs born to previously infected ewes.
 - a. Clinical findings include early papules followed by vesicles, which result in pustules and scabs on the lips, **muzzle**, and oral cavity in young animals. Lesions are painful, and animals are reluctant to eat. Debilitation due to starvation and secondary bacterial infection are the biggest dangers. Occasionally, ewes will have lesions on their udders.
 - b. Diagnostic plan and laboratory tests. The diagnosis is made based on clinical signs and the submission of a biopsy specimen for fluorescent antibody testing. Virus isolation may be performed on the exudate.
 - c. Therapeutic plan. Treatment includes supportive care, **force** feeding, and topical antibiotics.
 - d. Prevention. Vaccines are available for use in endemic **areas**. Lambs and kids should be vaccinated 2 weeks after weaning, and all show animals should be vaccinated 1 month before showing.
10. Equine **coital exanthema**. This is a sporadic venereal disease of horses that is characterized by necrotic, circumscribed lesions of the penis or **vulva/perineum**. Equine herpesvirus **type 3** infects animals, usually through breeding.
 - a. Clinical findings. Lesions are initially vesicular but rapidly progress to a dry pustule and ulceration stage.
 - b. Diagnostic plan. The diagnosis is confirmed by virus isolation from early vesicular lesions.

- c. Therapeutic plan. There is little effect on fertility, but secondary bacterial infections may occur. Affected stallions are treated with local application of antibiotic or steroid creams.

11. Systemic viral diseases with cutaneous manifestations
 - a. Bovine viral **diarrhea/mucosal** disease (see Chapter 1)
 - b. Malignant catarrhal fever (see Chapter 1)
 - c. Bluetongue (see Chapter 1)
 - d. Infectious bovine rhinotracheitis (see Chapter 6)
 - e. Foot and mouth disease (see Chapter 1)
 - f. Scrapie (see Chapter 11)
 - g. Border disease
 - h. Vesicular exanthema (see Chapter 1)
 - i. Vesicular stomatitis (see Chapter 1)

V. BACTERIAL SKIN DISORDERS

Folliculitis and furunculosis

1. Patient profile. These conditions are most commonly presented in horses and goats.
2. Definitions
 - a. Folliculitis—inflammation of the hair follicle
 - b. **Furunculosis**—an inflammatory process that breaks through the hair follicle and extends to surrounding dermis and **subcutis**
 - c. Carbuncle—coalescence of multiple areas of furunculosis in horses causing focal induration and fistulous tracts
3. Etiology. The major causative agents are the **Staphylococcus** species. **Coagulase**-positive staphylococci that are pathogenic in large animals include **S. aureus**, **S. intermedius**, and **S. hyicus**. Other agents include *Streptococcus* species, **Corynebacterium pseudotuberculosis** (Canadian **horsepox** or contagious acne), **Corynebacterium equi**, and *Dermatophilus congolensis* (see V C, E). In goats, causative agents include *Staphylococcus* species, **Actinomyces pyogenes**, and **Pseudomonas aeruginosa**.
4. Pathogenesis. Predisposing factors to folliculitis or furunculosis are mechanical trauma, insect bites, and moisture, combined with poor sanitation.
5. Specific conditions
 - a. Staphylococcal folliculitis. Initially, erect hairs may be found overlying a 2-mm to 3-mm papule. Lesions regress or progressively enlarge to 6–10 mm in diameter and develop a central ulcer that discharges serosanguinous material and becomes encrusted. During healing, the lesion flattens and is accompanied by alopecia and scaling, resembling ringworm.
 - b. Staphylococcal furunculosis. This disorder presents as a combination of nodules, draining tracts, ulcers, and crusts. Large lesions are edematous plaques that resemble urticaria. Scarring, leukoderma, and leukotrichia may follow.
 - c. Pastern folliculitis (greasy heel complex). Lesions are found on the caudal aspect of pasterns or fetlocks of horses and may be caused by a variety of organisms singly or in **concert**.
6. Diagnostic plan. The diagnosis is based on a complete history, a physical examination, direct smears and a bacterial culture of the lesions, and a skin biopsy.
7. Laboratory tests
 - a. A bacterial culture may reveal organisms in pure culture, but the interpretation may be difficult because of the normal occurrence on skin of some organisms (e.g., *Staphylococci*).

- b. A tissue biopsy reveals perifolliculitis with both folliculitis and furunculosis. There are extensive tissue eosinophilia accompanying furunculosis. Gram-positive bacteria may be visible.
8. Therapeutic plan
 - a. Mild cases often resolve spontaneously.
 - b. Severe cases require topical cleansing, drying, and antibacterial therapy (aqueous iodophors or chlorhexidine daily for 5–7 days, then twice weekly until resolved). Systemic antibiotics are necessary in severe progressive cases (procaine penicillin G 22,000 IU/kg twice daily for 7–10 days). Many coagulase-positive staphylococci are resistant to penicillin but are sensitive to erythromycin, trimethoprim-sulfamethoxazole, and aminoglycosides. Long-acting oxytetracycline (10–20 mg/kg) may be used if daily treatment is impractical.
9. Prognosis. Although healing is usually complete in 7–10 days, severe cases may not respond to treatment.

B. ulcerative lymphangitis

1. Patient profile. This sporadic disease is seen mainly in horses and is often associated with heavy populations of horses.
2. Clinical findings
 - a. **Lesions** occur on the lower limbs of the horse, particularly of the fetlock and seldom above the hock. Lesions begin as hard, fluctuant nodules, which abscess, ulcerate, and develop draining tracts of creamy green pus. Individual ulcers heal in 2 weeks, but new lesions develop.
 - b. Regional **lymphatics** are often corded, but there is no obvious lymph node involvement.
 - c. **Limb** edema and subsequent scarring is common.
3. Etiology. *Corynebacterium pseudotuberculosis* is most commonly isolated (a gram-positive pleomorphic rod). Other isolates include: *Staphylococcus* species, *C. pyogenes*, *Rhodococcus equi*, *Pasteurella haemolytica*, *Fusobacterium necrophorum*, and *Actinobacillus equuli*. Infections usually arise from wound contamination and are more prevalent in stabled animals where there are crowded conditions and poor hygiene.
4. Diagnostic plan. The diagnosis is based on history, a physical examination, and direct smears and a culture of the lesions.
5. Differential diagnoses include glanders and other bacterial causes of lower limb cellulitis.
6. Therapeutic plan. Hydrotherapy, exercise, surgical drainage, and high doses of penicillin (20,000–80,000 IU/kg, twice daily) for prolonged periods (more than 30 days) should be used.
7. Prognosis. The prognosis is poor for horses with significant scarring because recurrences are common.
8. Prevention. Good management practices provide the best means of control. Improving hygiene and decreasing population pressures is recommended.

C. Udder impetigo

1. Etiology. This condition in cattle is caused by *Staphylococcus aureus*. The infection is spread by the milker's hands and is zoonotic.
2. Clinical findings. Infection results in multiple pustules on the teat and udder skin. Cattle experience discomfort and a decrease in herd milk production as the disease progresses through the herd.
3. Therapeutic plan. Treatment is with systemic antibiotics or antibiotic disinfectant creams.

4. Prevention. Good sanitation is necessary for control (e.g., udder and milking machine disinfection, the use of latex gloves for milking personnel). An autogenous bacterin produces immunity for approximately 6 months in problem herds.

D Bacterial abscesses are very common in domestic animals following trauma and infection of deep tissues with staphylococci and a variety of other opportunistic organisms.

1. Chronic deep-seated pectoral abscesses (pigeon fever, pigeon breast, Wyoming strangles)
 - a. **Patient** profile. Adult horses are affected in the southwestern United States primarily in the autumn. Prevalence may be associated with heavy rainfall or high insect populations.
 - b. Clinical findings. Abscesses develop deep in the superficial musculature (most commonly in the pectoral region) and may measure 10–20 cm. The horse shows signs of fever, lameness, depression, anorexia, local edema, and pain with the rupture of the abscess within 2–4 weeks of identification.
 - c. Etiology and pathogenesis. *C. pseudotuberculosis* is the causative organism. It is hypothesized that the organism is transmitted via insect bites.
 - d. Diagnostic plan and laboratory tests. Diagnosis is based on clinical findings, characteristics of the purulent discharge, and the culture results.
 - e. Therapeutic plan. Antibiotics (penicillin at 20,000 IU/kg intramuscularly, twice daily) are used following surgical drainage.
2. **Caseous lymphadenitis (CLA)**
 - a. Patient profile. CLA is a common disease of adult sheep and goats that has a worldwide distribution.
 - b. Clinical findings. The disease presents as abscessed lymph nodes (subcutaneous nodules) that eventually break open and drain a thick, green pus.
 - (1) Abscesses are most common on the head and neck.
 - (2) Abscesses may also be found in the abdominal cavity and cause chronic weight loss (this manifestation is called visceral CLA or thin ewe syndrome).
 - (3) Abscesses may occur in other locations with clinical findings specific to the location (e.g., ataxia, pneumonia, renal disease).
 - c. Etiology and pathogenesis. *C. pseudotuberculosis* (biotype ovine/caprine) is the causative organism.
 - (1) Direct or indirect transmission of the organism occurs through contamination of skin wounds. Shearing or trauma allows the colonization of subcutaneous tissue and the abscessation of local lymph nodes. The organism persists in chronically draining lesions and in the soil.
 - (2) The visceral form evolves from hematogenous dissemination from the superficial lymph nodes. The visceral form may exist independent of the subcutaneous form.
 - d. Diagnostic plan and laboratory tests. For the subcutaneous form, the demonstration of abscessed lymph nodes with a high herd prevalence is highly suggestive of CLA. A microbiologic culture is diagnostic. Serology [e.g., enzyme-linked immunosorbent assay (ELISA)] identifies exposed animals but does not have good specificity.
 - e. Therapeutic plan
 - (1) Surgical drainage is the usual treatment of choice for individual animals but is often not attempted because locally abscessed lymph nodes cause few clinical problems. Lancing abscesses will only serve to contaminate the environment. Any surgical intervention must be linked with the isolation of the animal.
 - (2) Antimicrobials (e.g., penicillin, tetracycline) often fail because of the protected environment of the organism within thick-walled abscesses.
 - f. Prevention. The principles of prevention include:
 - (1) Culling infected sheep or goats (identified by superficial palpation)
 - (2) **Disinfecting** all common implements or tools (e.g., shears)
 - (3) Not purchasing animals from contaminated herds or flocks

(4) Vaccinating animals with commercial toxoids or bacterins

E. Actinomycotic infections

1. Actinomycosis (lumpy jaw; see Chapter 13 II B)
 - a. Patient profile. Actinomycosis is seen in adult cattle and sometimes in calves.
 - b. Clinical findings. A bony enlargement over the mandible or maxilla is seen and may cause stertorous breathing or pressure necrosis of the skin. These abscesses eventually rupture, discharging a thick, clear, yellow pus containing small yellow-white sulfur granules. Emaciation and death eventually ensue because of the animal's inability to eat.
 - c. Etiology and pathogenesis. The causative organism is *Actinomyces bovis*, which enters the subcutis through abrasions in the mouth.
 - d. Therapeutic plan. In animals with advanced lesions, treatment is disappointing. Recurrence is common.
 - (1) Early treatment consists of sodium iodide (1 g/12 kg body weight given intravenously once and repeated in 14 days).
 - (2) Penicillin (22,000 IU/kg intramuscularly twice daily for 7 days) is efficacious if used before bony lesions develop.
2. Dermatophilosis (streptothricosis, rainscald)
 - a. Patient profile. This disease may affect up to 80% of a herd or group of animals. It is most common in horses but can be seen in cattle, sheep, and goats. This condition is potentially zoonotic.
 - b. Clinical findings. Early signs of infection include **follicular** and nonfollicular papules and pustules that rapidly coalesce and become exudative. These lesions cause groups of hairs to become **crusted** and matted together. Proximal portions of matted hair embedded in exudate results in the "paintbrush effect." White-skinned areas (e.g., the muzzle, distal limbs) are more severely affected and may exhibit severe erythema as a result of photodermatitis.
 - (1) Distribution of lesions. Lesions occur primarily over the dorsum but are also seen on the scrotum, perineum, udder, and distal limbs.
 - (a) The disease affects the ears and tail base of goat kids and causes **wool-mats** in sheep (lumpy wool disease).
 - (b) The disease on coronary bands and carpi of sheep may be termed **strawberry foot rot**.
 - (c) Exudative crusted lesions over the rump and top line may be referred to as **rainscald**.
 - (d) Lesions involving the **distal limbs** (i.e., pasterns, coronets, heels) may be called **greasy heel**, **scratches**, or **mud fever**. Distal limb lesions may be associated with edema, pain, and lameness.
 - (2) Epithelial defects. Removal of hair tufts from larger lesions over the back exposes ovoid, ulcerated, hemorrhagic, purulent, and painful epithelial defects. Active lesions contain thick, creamy, yellowish or greenish pus that adheres to the skin surface and undersurface of crusts. Acute, active lesions are **painful** but not pruritic.
 - c. Etiology and pathogenesis
 - (1) Causative agent. *Dermatophilus congolensis* is a gram-positive, facultative, anaerobic actinomycete. Clinically silent but chronically infected animals become a source of infection under favorable climatic conditions.
 - (2) Predisposing factors
 - (a) Skin damage. Crucial to development of dermatophilosis is skin damage (e.g., biting flies and arthropods, prickly vegetation, maceration).
 - (b) Moisture, such as that occurring with heavy rainfall, is essential in the pathogenesis by allowing for the release of infective, motile zoospores.
 - (c) Concurrent **diseases** and immunosuppression predispose animals to disease.

- (d) Other putative predisposing factors include high ambient temperature, high humidity, poor nutrition, poor hygiene, and stress.
- d. Diagnostic plan. A history and physical examination should provide the diagnosis, which is confirmed by laboratory tests.
- e. Laboratory tests
 - (1) Stains. The diagnosis is confirmed by direct smears of pus or **saline-soaked** minced crusts stained with new methylene blue stain, Diff-Quik, or Gram stains.
 - (a) Appearance. *D. congolensis* appears as fine-branching, multiseptate hyphae that divide transversely and longitudinally to form cuboidal packets of coccoid cells arranged in two to eight parallel rows within branching filaments (i.e., a railroad-track appearance). In the chronic or healing phase of the disease, direct smears are rarely positive.
 - (b) Negative results. If a dermatophilus preparation is negative in a suspect case, the diagnosis should not be ruled out without submitting the crusts for microbiology.
 - (2) Culture is performed on blood agar in a microaerophilic atmosphere. *D. congolensis* is cultured only from integument and crusts in affected animals.
 - (3) A skin biopsy reveals folliculitis, intraepidermal pustular dermatitis, superficial perivascular dermatitis, and intracellular edema of keratinocytes. Surface crusts are composed of **alternating** layers of keratin and leukocytic debris. *D. congolensis* organisms are present in leukocytic debris and hair follicles.
- f. Differential diagnoses include dermatophytosis, staphylococcal folliculitis and furunculosis, zinc-responsive dermatosis, and pemphigus **foliaceus**.
- g. Therapeutic plan. Healing is characterized by dry crusts, scaling, and alopecia (ringworm-like lesions).
 - (1) Systemic therapy is needed for animals with severe, generalized, or chronic infections. Systemic antibiotic regimens include 22,000 IU/kg of procaine penicillin twice daily for 7 days.
 - (2) Crust removal and topical treatment will suffice for patients with less generalized dermatitis. Topical solutions should be applied as total body washes, sprays, or dips for 3–5 consecutive days and then weekly until healing. Treatments include iodophors (e.g., povidone iodine shampoo), 2%–5% lime sulfur, 0.5% zinc sulfate, 0.2% copper sulfate, or 1% potassium aluminum sulfate.
- h. Prognosis. Most lesions regress in 4 weeks if the animals are kept dry.
- i. Prevention. Previously infected animals do not develop significant immunity, so reinfection is always a possibility.
 - (1) Owners should be instructed to improve hygiene, nutrition, and management practices.
 - (2) Arthropod and insect control measures should be employed, and affected animals should be isolated.
 - (3) Measures should be taken to limit mechanical skin trauma.

F. Erysipelas

1. Patient profile. Erysipelas is a disease of growing or young adult swine (particularly young sows) and has a global distribution. Erysipelas is a **zoonotic** disease and people handling infected animals or carcasses are at increased risk of infection.
2. Clinical findings. There are two clinical expressions of the disease.
 - a. The acute **form** involves an acute septicemia that is characterized by sudden death or high fevers, depression, anorexia, and, in a subset of animals, classic diamond-shaped, **hyperemic** plaques.
 - (1) These plaques usually follow the other signs of septicemia and are most prominent on the underside of the pig or on the ears. The skin lesions are

often palpable before they can be visualized, and if the animal lives, these lesions may go on to coalesce.

- (2) Disease **progression**. Pigs affected by the acute form die after exhibiting signs of dyspnea, cyanosis, and diarrhea. The mortality rate may be high (75%), but the mortality rate depends on the pathogenicity of the strain of the organism and the immunity of the herd. With pigs that recover, the skin lesions become dry, hard, and eventually slough.
- b. A chronic form of the disease manifests as arthritis, endocarditis, or both. Lameness and stiffness is evident, particularly in the knee, elbow, hip, hock, and stifle. Animals may suffer chronic ill-thrift or may die suddenly due to the heart lesions.
3. Etiology and pathogenesis. *Erysipelothrix rhusiopathiae* is the causative organism. There are many different serotypes with varying degrees of virulence. The organism persists in the environment, which is contaminated from the feces of carrier pigs. Susceptible animals are most likely infected via the oral route.
4. Diagnostic plan and laboratory tests. Classic lesions, herd epidemiology, and necropsy findings (including bacterial culture of affected tissues) lead to the diagnosis.
5. **Differential** diagnoses. Diseases with similar clinical expressions include hog cholera and **salmonellosis**. Necropsy results rule out these diseases.
6. Therapeutic plan. The treatment of choice for acutely affected pigs is penicillin (50,000 IU/kg intramuscularly, once daily for 3 days). Pigs with the chronic form of the disease do not respond well to treatment.
7. Prevention. Vaccination is somewhat effective in controlling the disease, particularly if a booster is given 30 days after the initial vaccination. Vaccination should be coupled with increased attention to hygiene and biosecurity measures aimed at limiting access to facilities by pigs, personnel, feral animals, and equipment.

G. Exudative epidermitis (greasy pig disease, exudative dermatitis)

1. Patient profile. This disease is seen worldwide. Entire litters of nursing piglets may be affected. Usually, this disease is of less clinical importance as animals get older.
2. Clinical finding
 - a. In young piglets, there is marked skin reddening with seborrhea and cutaneous pain. There is anorexia, depression, and dehydration leading to death despite therapy.
 - b. Older piglets have the characteristic greasy pig expression with thick, brown seborrheic scabs that are most prominent around the head and face. The skin is thickened, and there is a characteristic odor. Piglets usually go on to recover if treated.
3. Etiology. The causative agent is *Staphylococcus hyicus* and is possibly spread by carrier sows. The organism gains entrance through abrasions, most often around the face and head. However, the disease is extremely infectious.
4. Diagnostic plan and laboratory tests. Diagnosis depends on clinical signs combined with a culture of the skin exudate and slide agglutination with *S. hyicus* antisera.
5. Therapeutic plan and prognosis. The organism is sensitive to most antibiotics (excluding sulfa drugs) if intervention occurs within 2 days of the development of clinical signs. After this time, the prognosis is poor.
 - a. Procaine penicillin (20,000 IU/kg, intramuscularly daily for 3 days) has shown good efficacy.
 - b. **Local** treatment with mild disinfectant or soapy rinses is **also** of value.
6. Prevention. Improvements in hygiene and biosecurity are recommended. After an outbreak, the premises should be disinfected and left vacant for 10–14 days. All

implements used in treating piglets (e.g., pliers, tattooing equipment) should be thoroughly disinfected.

VI. FUNGAL DERMATOSES

A. Introduction

1. Definitions
 - a. Mycosis is a disease caused by a fungus.
 - b. Dermatophytosis is the infection of keratinized tissues (i.e., nail, hair, and stratum corneum) by species of *Microsporum*, *Trichophyton*, or *Epidermophyton*.
2. Hosts. Dermatophytes may be transmitted from animals to man and vice versa. Zoophilic fungi prefer animals as hosts. These fungi rarely cause acute inflammation in animals but often cause severe inflammatory reactions when they invade humans.

B. Superficial mycoses. Dermatophytosis (dermatomycosis, ringworm, tinea, girth itch) occurs most commonly in young animals.

1. Clinical findings
 - a. Horses. Lesions are common on the saddle and back areas, thorax, head, and shoulders. Lesions may be discrete, small (2–3 cm), round areas of erect hairs that progress to alopecia. Also, there may be generalized scaling (seborrhea **sicca**) without alopecia. Pruritus and pain vary from severe to absent. Lesions may be limited to the posterior pastern region (these lesions may be known as scratches, mud fever, greasy heel-like) and may wax and wane with stress, local irritation, moisture, and unsanitary conditions.
 - b. Cattle. Affected areas are typically characterized by raised, grey, crusted lesions that begin as discrete areas of alopecia but rapidly become confluent. The neck, face, and head are most commonly affected, and the lesions are not pruritic. Lesions are described as "asbestos-like" scales.
 - c. Pigs. Areas that are commonly affected include the topline and sides of the animal. Lesions appear as progressive rings of alopecia. The interior of the rings is scabby and **alopecic**.
 - d. Sheep and goats
 - (1) In sheep, the head is the most common site of involvement, with discrete, circular areas of alopecia. These lesions are covered with a grey **crust**.
 - (2) In goats, there may be whole-body involvement
2. Etiology and pathogenesis
 - a. Etiology. Microsporum species and *Trichophyton* species are the most frequently isolated animal dermatophytes.
 - (1) Genera are divided into three groups on the basis of natural habitat:
 - (a) **Geophilic** organisms inhabit the soil (e.g., *Microsporum gypseum*, *Microsporum nanum*, *Trichophyton verrucosum*, *Trichophyton mentagrophytes*).
 - (b) **Zoophilic** organisms prefer animals to humans (e.g., *Microsporum canis*, *Trichophyton equinum*).
 - (c) **Anthropophilic** organisms prefer humans to other animals (e.g., *Microsporum audouinii*).
 - (2) Predisposing factors for development of ringworm include:
 - (a) Age (young animals are most susceptible)
 - (b) Immunity (e.g., immunosuppression, reduced T-cell function)
 - (c) Environment (e.g., contamination, crowding, high humidity, poor ventilation, darkness)
 - (d) Poor condition (e.g., poor nutrition, debilitating diseases)
 - b. Pathogenesis

- (1) Transmission. The causative organisms are transmitted by direct contact with infected animals or fomites. The incubation period post-contact is 1–6 weeks, and fungal spores remain viable for years.
- (2) Infection. Dermatophytes do not invade living tissue. The organisms invade the hair shaft, causing breaking and alopecia. These organisms elaborate toxins (irritants) or allergens that enter the dermis and evoke an inflammatory response.
- (3) Host. Dermatophytes are adapted to survive on the skin of a particular host but cause violent responses in a host that is not adapted to its presence (e.g., zoophilic dermatophytes in humans).
- c. Diagnostic plan. The condition is most commonly diagnosed by clinical findings and confirmed by laboratory tests.
- d. Laboratory tests. For confirmation, microscopic examination of the hair and surface debris and a fungal culture are necessary.
 - (1) Microscopic examination. For **potassium hydroxide (KOH)** preparation, several drops of 20% KOH should be applied to a glass slide onto which hair and keratin have been placed. This should be heated gently for 20 seconds and then examined microscopically for arthrospores on or in hair shafts.
 - (2) Fungal culture media include:
 - (a) **Dermatophyte test medium (DTM)** plus 2 drops of injectable B vitamin complex
 - (b) Sabouraud's dextrose agar with cycloheximide and chloramphenicol
 - (3) A Wood's lamp test to produce fluorescence can be attempted, but this test usually is negative in large animals with dermatophytosis.
 - (4) Skin biopsy. If a skin biopsy is employed, histopathologic findings are variable but include:
 - (a) Perifolliculitis, folliculitis, and furunculosis
 - (b) Superficial perivascular dermatitis
 - (c) Intraepidermal vesicular or pustular dermatitis
 - (d) **Septate** fungal hyphae and spherical to oval conidia within the surface keratin and crust, within the hair follicles, or in and around the hairs
- e. Differential diagnoses include staphylococcal dermatitis, dermatophilosis, demodicosis, zinc-responsive dermatosis (in ruminants), and pemphigus foliaceus (in horses).
- f. Therapeutic plan
 - (1) Safety measures. Caution should be exercised during treatment because the disease is zoonotic.
 - (a) Animals that test negative for the disease should be **isolated**, and all in-contact animals should be treated.
 - (b) The environment and fomites should be disinfected, and all infectious materials (e.g., crusts, hair, bedding) should be disposed of.
 - (c) The environment can be treated with 5% lime sulfur, 5% sodium hypochlorite, 5% formalin, 3% **captan**, or 3% **cresol**.
 - (2) Topical antifungal agents. Treatments may differ depending on the species, label specifications, and animal use. Some topical antifungal agents include:
 - (a) **2%–5% lime sulfur**
 - (b) 3% **captan** (1–2 ounces of a 50% powder in 1 gallon water)
 - (c) **Iodophors**
 - (d) 0.5% sodium hypochlorite once daily for 5 days as a body spray or dip, then once weekly until clinical cure is evident
 - (e) **1%–5% thiabendazole** solution applied topically once every 3 days
 - (3) Systemic therapy is controversial but may be effective and **necessary** in some instances. Systemic treatments include:
 - (a) Oral thiabendazole (50–100 **mg/kg**)
 - (b) Sodium iodide (10%–20%) at 1 **g/14 kg** (repeat in 7 days)
 - (c) **Griseofulvin** is expensive, and its use is probably not warranted under usual circumstances.

(i) Cattle: 5–7.5 **mg/kg** orally for 7–10 days

(ii) Swine: 1 **g/100 kg/day** orally for 7–40 days

(iii) Horses: 5–10 **mg/kg/day** orally for 7–10 days

- g. Prognosis. Most lesions regress spontaneously in 2–3 months, particularly if the nutritional plane is good and the animal is exposed to sunlight. Dermatophytosis is normally self-limiting with spontaneous remission in 1–4 months.
- h. Prevention. Control depends on reducing contagion to the environment, other animals, and humans while waiting for spontaneous remission.

C. Subcutaneous mycoses

1. Equine sporotrichosis
 - a. Clinical findings. Lesions are usually confined to the limbs, although occasionally they may be found on the upper body (i.e., shoulder, hip, perineal regions). Lesions are hard, subcutaneous nodules that develop progressively along lymphatics that may become corded. Large nodules may **abscess**, ulcerate, and discharge a small amount of thick, brownish red pus or **serosanguinous** fluid.
 - b. Etiology and pathogenesis. The etiologic agent is *Sporotrichum schenckii*, a dimorphic fungus. This organism is a soil and vegetation inhabitant that has a worldwide distribution. *S. schenckii* enters the host via wound contamination (e.g., puncture wounds from thorns, wood splinters, bites).
 - c. Diagnostic plan. Diagnosis is primarily based on history and physical examination findings; a skin biopsy may also be useful.
 - d. Laboratory tests
 - (1) A skin biopsy shows hyperplastic perivascular dermatitis early in the course of the condition. Later, there is diffuse or nodular dermatitis. *S. schenckii* present as round to oval cells producing buds ranging from 3–6 μm in diameter. Classic "cigar buds" are less commonly observed. *S. schenckii* is often impossible to find in histologic sections.
 - (2) Direct culture from exudate or tissues is reliable because the organism grows readily on Sabouraud's agar.
 - e. Differential diagnoses include bacterial and fungal granulomatous disorders, such as **glanders** and ulcerative lymphangitis.
 - f. Therapeutic plan. This is a **zoonotic** disease, so caution should be exercised when handling infected animals; care should be taken to avoid contact with discharge from nodules. Therapy is iodide administration.
 - (1) Preparations
 - (a) Oral inorganic iodides. A 20% solution of sodium iodide may be used intravenously at 40 **mg/kg** for 2–5 days. This is followed by potassium (orally, once daily) until the lesions regress.
 - (b) Potassium iodide may be used at 1–2 **mg/kg** orally twice a day for 1 week, then decreased to 0.5–1 **mg/kg** orally once a day until 3–4 weeks after the lesions have resolved.
 - (2) Side effects
 - (a) Some horses may exhibit iodism (scaling, alopecia, depression, anorexia, fever, coughing, lacrimation, nasal discharge) with therapy. If side effects develop, the dose should be reduced, or treatment should be temporarily discontinued.
 - (b) Systemic iodides may cause abortion in pregnant mares.
2. **Phycomycosis (pythiosis, Florida horse leeches)**
 - a. Patient profile. This condition affects horses in tropical or subtropical climates. In North America, the disease is most common along the Gulf of Mexico coast.
 - b. Clinical finding. The lower limbs, abdomen, neck, and head may be affected by ulcerated, necrotic, yellow-gray, draining tracts. These lesions are pruritic and may appear similar to exuberant granulation tissue. Necrotic, organized cores within the masses give the disease the common name leeches.

- c. Etiology and pathogenesis. Several fungi within the class *Phycomycetes* may be responsible for the condition. The most common organisms are *Hyphomycetes destruens* and *Entomophthora coronata*. These organisms may gain entrance to the horse through sites of trauma or skin barrier breakage (e.g., due to moisture).
- d. Diagnostic plan and laboratory tests. The diagnosis is confirmed by the histopathology of the lesion. A preparation cleared with KOH reveals hyphal elements.
- e. Therapeutic plan
 - (1) Surgical debridement of lesions combined with amphotericin B injections has proven to be the most effective treatment. Amphotericin B (150 mg gradually increasing to 400 mg/450 kg) is nephrotoxic, so kidney function should be monitored via blood urea nitrogen (BUN) levels during treatment. Treatment may be necessary for up to 30 days.
 - (2) Dimethylsulfoxide (DMSO) also has been beneficial when applied locally in certain cases.
- f. Prevention. Good hygiene and dry skin should be maintained when possible.

VII. PARASITIC DISEASES

A. Introduction. Parasitic diseases cause animal suffering through annoyance, irritation, pruritus, disfigurement, and secondary infection. There are also economic losses from decreased weight gain and milk production, hide damage, wool loss, death, and the financial burden of diagnostic, therapeutic, and preventative programs. Treatment of ectoparasitism is a complex issue because of:

1. Regional differences in availability and control of therapeutic agents
2. Species and age differences among hosts
3. Meat and milk withdrawal time variability
4. Safety issues in pregnant animals

B. Mange

1. **Psoroptic** mange (sheep scab, ear mange)
 - a. Patient profile and history. This intensely pruritic skin disease is seen in cattle, sheep, goats, and horses.
 - b. Clinical finding
 - (1) Cattle. Early lesions form as papules on the withers, neck, and tailhead. As these papules coalesce, hair is lost, and the skin becomes wrinkled and thickened. There is intense pruritus.
 - (2) Horses may appear clinically normal or exhibit variable head shaking, ear scratching, and rubbing. Long-haired areas of the body (e.g., mane, tail) may also be affected, resulting in rubbing, thickened skin, and hair loss.
 - (3) Sheep. Papules occur most commonly on the thorax. These lesions ooze serum, coalesce into scabs, and mat the fleece. The fleece is lost as the animals become itchy. Flocks may be less negatively affected if nutrition and management is good.
 - (4) Goats. Although there may be whole-body involvement, the condition is most commonly one of scabs on the external ear canal. Lesions may range from mild scabs to large proliferative-looking scabs occluding the ear canal.
 - c. Etiology and pathogenesis. *Psoroptes* species mites are nonburrowing but do cause skin excoriation. These parasites feed on both blood and tissue fluids. Transmission is via direct and indirect contact.
 - (1) *P. ovis* is the only mite that affects sheep, but it may also be found on other species (e.g., cattle).

(2) *P. equi* occurs on horses.

(3) *P. cuniculi* is the ear mite of various animals.

- d. Diagnostic plan and laboratory tests. Routine skin scrapings provide the diagnosis. Also, otoscopic examination may be performed to detect ear mites, followed by a microscopic examination of the material collected from deep within the ear canal.
 - e. Differential diagnoses include sarcoptic mange, chorioptic mange, psorergatic mange, lice, keds, and flybite dermatitis.
 - f. Therapeutic plan
 - (1) Sheep may be treated with a whole-body dip of approved ascaricides (e.g., 0.01% diazinon, 0.5% malathion, 0.06% coumaphos). Retreatment according to label directions is necessary.
 - (2) Cattle. Ivermectin (0.2 mg/kg subcutaneously) is efficacious for mites in cattle.
 - (3) For horses or goats with ear mites, commercial dog and cat ear mite preparations are effective after a thorough cleaning of the ear with benzene hexachloride.
 - g. Prevention. Psoroptic mange may be a reportable disease in many countries; therefore, notification of federal authorities is indicated when the disease is suspected. Prevention of the disease in sheep requires a whole-body treatment (i.e., dipping) at least twice yearly. Mites survive off of the host for 2 weeks, so it is important to leave the environment unpopulated for this time period.
2. Sarcoptic mange (barn itch, scabies)
 - a. Patient profile and history. This parasitic condition is most common in pigs but all species of animals may be affected. The main complaint from owners is an intense pruritus in the animal. The most susceptible animals are those that are thin and in overcrowded conditions. The condition is most prevalent in the winter stabling period.
 - b. Clinical findings include an intense pruritus of face, neck, shoulders, and rump. Nonfollicular papules, crusts, excoriations, and alopecia are seen.
 - c. Etiology and pathogenesis
 - (1) Etiology. Severe, chronic sarcoptic mange is associated with poor feeding, management, and hygiene practices on the farm. The causative agents are the *Sarcoptes* species.
 - (2) Pathogenesis
 - (a) Infection and life cycle. These organisms tunnel through epidermis to feed on tissue fluids and possibly epidermal cells. The parasite life cycle is completed in 2–3 weeks. Mites are susceptible to drying and survive only a few days when separated from the host.
 - (b) Pruritus in scabies is caused by a hypersensitivity reaction to mite products. Piglets become infected from sows shortly after birth and develop the hypersensitivity reaction and pruritus 8–10 weeks later. The reaction is characterized by tissue eosinophilia, positive intradermal tests using mite antigen, and difficulty in detecting mites on skin scrapings.
 - d. Diagnostic plan. The history and a physical examination lead to a presumptive diagnosis because this parasite is the only mange mite that affects pigs. Response to therapy is useful for diagnosis in small groups of animals because mites are difficult to find. Laboratory testing confirms the diagnosis.
 - e. Laboratory tests
 - (1) Positive skin scrapings confirm the diagnosis, but mites can be difficult to find. Cerumen gathered from the ear canal and pinnae are the most consistent source of ear mites in swine.
 - (2) A skin biopsy shows a superficial, perivascular dermatitis with numerous eosinophils. Occasionally, mites are visible in parakeratotic scale crusts and subcorneal "tunnels."
 - f. Differential diagnoses include psoroptic mange, chorioptic mange, psorergatic mange, lice, keds and flybite dermatitis.

- g. Therapeutic plan. This mange is a reportable condition to federal authorities in many constituencies. Cross-infections between humans and animals occur.
 - (1) **Ivermectin** is the standard treatment, although insecticides may also be used.
 - (2) Dips should be applied at least twice in a 14-day interval. Several agents are available (e.g., 0.5% malathion, 0.03% lindane, 0.06% coumaphos, 0.5% methoxychlor, 2% lime sulfur). Insecticide sprays should also be used in the surrounding stable unless the stable can be depopulated for 3 weeks.
 - (3) Nutrition and the general health of the animals should be improved.
3. Chorioptic mange (leg mange, tail mange, symbiotic scab, **scrotal** mange, foot mange)
 - a. Patient profile. This is the most common type of mange in cattle and horses. Horses with feathered fetlocks (i.e., draft horses) are prone to the condition.
 - b. Clinical findings
 - (1) Horses exhibit pruritus of the lower legs by stamping, rubbing, and stepping. Close examination may reveal scabs and a greasy dermatitis. Chorioptic mite infestation may be a component of greasy heel.
 - (2) Cattle are affected by multiple small scabs along the udder, thighs, and perineum. In extreme cases, the coronet and muzzle may be affected. These lesions are not pruritic.
 - (3) Rams exhibit a **scrotal** dermatitis.
 - c. Etiology and pathogenesis
 - (1) Etiology. The major causative species of mites is *Chorioptes bovis*, which can infect most domestic animals. Chorioptic mites are surface-inhabiting parasites that feed on epidermal debris. They do not affect humans.
 - (2) Pathogenesis
 - (a) Transmission is by direct contact or via common equipment (e.g., combs, brushes).
 - (b) **Life** cycles, which span 3 weeks, are completed on the host. Mite populations are affected by the season and are heaviest at times of cooler temperatures and higher humidity. There is spontaneous disease regression in the summer because the mite population decreases.
 - (c) The mites cause an allergic dermatitis.
 - d. Diagnostic plan and laboratory tests. A combination of history, physical examination, and skin scrapings leads to the diagnosis. Mites may be demonstrated around the coronet and interdigitally during clinically inapparent periods.
 - (1) Microscopic findings. Chorioptic mites are easy to demonstrate. Live mites may move fairly rapidly through the microscopic field, so a mixture of one-part rotenone to three-parts mineral oil is recommended.
 - (2) A skin biopsy will show superficial perivascular dermatitis with numerous eosinophils. Mites are rarely found.
 - e. Therapeutic plan and prevention. Infected and in-contact animals should be treated.
 - (1) **Ivermectin** should be used at recommended dosages and repeated in 2 weeks.
 - (2) Other treatments include the total body application of 0.5% malathion, 0.5% methoxychlor, 0.06% coumaphos, or 0.25% crotoxyfos, bearing in mind labeled clearances for the species of interest.
 - (3) None of the treatments eliminate the infection from the herd or flock, and reinfections usually occur in subsequent years.
4. Psorergatic mange (Australian itch, itch mite)
 - a. Patient profile. Psorergatic mange is a condition of sheep that has a world-wide distribution.
 - b. Clinical findings. Affected animals rub and bite their flanks, thighs, and sides. Their wool becomes tattered and scaly. Usually only a small percentage of the flock is affected because young animals have not developed the disease yet and older animals may be the carriers.

- c. Etiology and pathogenesis. *Psorergates ovis* completes its entire life cycle on the sheep in 4–5 weeks. The mite is active only in the superficial layers of skin, causing a mechanical irritation initially and, usually, a hypersensitivity reaction over time. Mite numbers are highest in the winter months.
 - d. Diagnostic plan. The clinical signs of disease appear similar to other external parasites, such as sheep keds. For diagnostic testing, animals with the appearance of scurfy skin should be selected and their skin scraped for mites along the ribs or shoulder.
 - e. Therapeutic plan and prevention. The condition is difficult to eradicate, but ivermectin and common insecticides are used.
5. **Demodectic mange** This type of mange affects all domestic animals but is most significant in goats.
 - b. Clinical findings. Skin nodules (3 mm in size) are apparent on the face, neck, shoulders, and sides. Lesions are not pruritic and are more easily palpated than seen. These lesions form small caseated abscesses. There may be some minor alopecia and hyperkeratosis.
 - c. Etiology and pathogenesis. *Demodex* species may be considered normal skin residents. Demodectic mites live in hair follicles and sebaceous glands and are host-specific. It is thought that clinical demodicosis occurs in immunocompromised animals or animals with concurrent diseases (e.g., ringworm). In herd outbreaks, genetic predisposition or endogenous/exogenous causes of immunosuppression should be considered.
 - d. Diagnostic plan. History and physical examination findings are supported by skin scrapings of alopecic or scaly areas.
 - e. Laboratory tests
 - (1) Skin scraping. Affected areas should be squeezed firmly and scraped deeply until blood is drawn. Nodular lesions should be incised and manually evacuated to reveal exudate loaded with *Demodex* mites.
 - (2) A skin biopsy shows follicular distention with mites accompanied by granulomatous/eosinophilic perifolliculitis, folliculitis, or furunculosis.
 - f. Therapeutic plan. Ascaricides that are recommended for other mites may help control *Demodex* infection but will not be curative. **Ivermectin** (0.3 mg/kg administered by subcutaneous injection) has proven efficacious. A limited number of large, localized lesions in goats can be incised, expressed manually, and infused with **Iugols** iodine or rotenone in alcohol (1:3).
 - g. Prevention. Severely affected animals should be culled or not used for breeding stock because this disease is difficult to treat, and there is genetic predilection for disease susceptibility.

C Ked, louse, and tick infestation

1. Sheep keds
 - a. Patient profile and history. Sheep keds has a global distribution. Heaviest infestations are seen during cool, wet times (winter).
 - b. Clinical findings. The usual infestations are light to moderate, so clinical signs are limited to slight irritation and scratching. Self-trauma may cause wool damage. Heavier infestations result in ill-thrift and anemia. Keds are visible to the naked eye and measure 6–7 mm in length.
 - c. Etiology and pathogenesis. *Melophagus ovinus* (wingless fly), an obligate parasite, lives its entire life cycle on sheep (occasionally on goats), feeding on blood.
 - (1) The spread of this parasite is most likely by direct transmission. Females deposit single larvae and are not prolific (approximately 1 larva/week), so populations build slowly.
 - (2) Adult sheep gradually build up resistance to the parasite.
 - (3) Keds may also transmit the blue tongue virus.
 - d. Diagnostic plan. Keds are readily seen on physical examination if the wool is parted.

- e. Therapeutic plan. Shearing removes many keds, but these parasites are also susceptible to many ascaricides (e.g., organophosphates) and ivermectin.
2. Lice (**pediculosis**)
 - a. Patient profile. Any of the common domestic animals are susceptible to louse infestation.
 - (1) Heaviest infestations seem to occur on younger animals and those in the poorest condition.
 - (2) Animals unable to practice grooming are most often affected (e.g., stanchioned cattle).
 - (3) Lice infestation seems to occur more commonly in winter and is particularly common in goats. Lice populations are much higher in the winter because of lower skin, hair, or wool temperatures, overcrowding, poor nutrition, and the availability of moisture. High skin temperatures in the summer kill off lice; therefore, small populations survive only inside ears, between legs, and in tail switches.
 - b. Clinical findings. Affected animals suffer from skin irritation, a roughened hair coat, alopecia, and anemia. Lice may occur over the entire body in heavy infestations but are more common over the back, neck, brisket, tail head, and any long-haired part of the body.
 - c. Etiology and pathogenesis
 - (1) Etiology. Lice can be classified as sucking lice or biting lice. Within each category, species are host specific.
 - (a) Biting lice include *Damalinia bovis*, *Damalinia ovis*, *Damalinia caprae*, *Damalinia limbata*, and *Damalinia crassipes*.
 - (b) Sucking lice
 - (i) Cattle: *Hematopinus eurysternus*, *Haematopinus quadripertusus*, *Linognathus vituli*, *Solenopotes capillatus*
 - (ii) Sheep: *Linognathus pedalis*, *Linognathus ovillus*
 - (iii) Goats: *Linognathus stenopsis*
 - (iv) Horses: *Damalinia equi*, *Haematopinus asini*
 - (2) Pathogenesis
 - (a) The life cycle of lice is completed from egg to adult on the host and takes 2–4 weeks. Lice can survive for up to 7 days away from the host.
 - (b) Transmission is by direct contact or fomites.
 - (c) Sucking lice may cause anemia through blood removal. Biting lice mainly cause skin irritation because they feed on epithelium and cutaneous debris.
 - d. Diagnostic plan. Demonstration of lice and the eggs (nits) is definitive. However, careful examination is necessary to visualize the lice, particularly on a dark haircoat or in a dark environment. The hair should be parted and a flashlight used to look for the pale-colored (almost transparent) biting louse or the blue-grey sucking louse. Nits are attached to the hair shaft.
 - e. Therapeutic plan
 - (1) Products effective in horses, cattle, goats, sheep and swine include many organophosphates and synthetic pyrethroids. The efficacy of these products is enhanced by the removal of long hair (e.g., shearing in sheep, clipping in cattle). Resistance to these products can occur if used singly over time.
 - (2) **Ivermectin** may also be used (200 µg/kg) and is extremely effective against sucking lice. It is also reported to be effective against biting lice. All animals in the group should be treated, although lice may only be seen on one animal (old or sick).
 - f. Prevention. Improved hygiene and nutrition are important control measures. The environment should be kept clean, and animal crowding should be decreased.
3. Ticks. Heavy infestations of ticks limit weight gains and productivity in animals because of irritation and annoyance.

- a. Patient profile. Ticks are parasitic on all domestic animals and have a global distribution.
- b. Clinical findings. Ticks may be found in the ear canal or on the body.
 - (1) Anemia. Many tick species are bloodsuckers and can cause profound anemia.
 - (2) Paralysis or other central nervous system (CNS) signs may occur because of a toxin elaborated by the salivary glands of female ticks.
- c. Etiology and pathogenesis
 - (1) Etiology. The most common ticks in North America are the *Dermacentor*, *Boophilus*, *Amblyomma*, *Otobius*, and *Ixodes* species.
 - (2) Pathogenesis
 - (a) Life cycles are variable. Some species are obligate parasites of a single host, whereas other species have various and successive hosts. Generally, eggs are laid in the soil, and larvae attach to passing hosts. The larvae mature to adults. Females feed on blood and drop to the ground to lay eggs.
 - (b) The blood-feeding phase of the cycle promotes infection with various bacterial, viral, and rickettsial diseases that are dependant on the location and epidemiology of the disease.
 - (c) Other diseases found in North America related to tick infestation include:
 - (i) **Babesiosis**
 - (ii) **Tularemia**
 - (iii) **Anaplasmosis**
 - (iv) **Caseous lymphadenitis**
 - (v) **Epizootic bovine abortion**
 - (vi) **Lyme disease**
- d. Diagnostic plan. Diagnosis is based on observation and the identification of the tick.
- e. Therapeutic plan and prevention
 - (1) Insecticides. Treatment with insecticides is the usual course of action. Various agents (such as pyrethroids, organophosphates, or ivermectin) may be used through sprays, dips, or powders. A regular, consistent program must be employed. However, resistance may develop and can be a problem in any control program.
 - (2) Control is difficult because of the life cycle of the tick off of the animal and its ability to live for extended periods of time away from its hosts. Consequently, not many tick control programs have been successful.

D. Fly-related dermatoses

1. **Hypoderma** infestation (warbles, grubs). This infestation, if heavy, may worry the cattle and cause production losses. Other significant losses are caused by carcass and hide depreciation and the cost of control programs.
 - a. Patient profile and history. Hypoderma infestation is most common in beef cattle, although it may be seen in dairy cattle raised in feedlot conditions and occasionally in horses in poor condition raised in close proximity to cattle. Young cattle are most severely affected.
 - b. Clinical findings
 - (1) Small numbers of painful subcutaneous nodules, each with a breathing pore, are seen over the withers of young cattle in the spring. Occasionally, nodules can be extremely numerous.
 - (2) Other clinical findings are associated with reactions to larvae, interrupted migration, or aberrant migration of larvae. All of these secondary reactions are relatively infrequent.
 - (a) Bloat. An inflammatory reaction may occur around larvae in the submucosa of the esophagus, causing esophageal obstruction and a buildup of rumenal gases.

- (b) Paralysis results from an inflammatory reaction surrounding dead larvae in the spinal column (in cattle and horses).
- (c) Intracranial myiasis (in unusual hosts such as horses, goats, humans, or sheep). Larvae may migrate aberrantly and do not complete their life cycle but cause neurologic signs if an inflammatory reaction to dying larvae occurs intracranially.
- (d) Anaphylactic reaction. This reaction is associated with the rupture of L₃. The therapeutic destruction of L₂ has also been recorded as a cause of anaphylactic reaction (in cattle).
- c. Etiology and pathogenesis
 - (1) Etiology. Hypoderma **bovis** and Hypoderma **lineatum** are the adult flies of the pathogenic larvae.
 - (2) Pathogenesis
 - (a) The adult flies are active in the summer and fix eggs to hairs on the legs of cattle. Larvae that hatch penetrate the skin and migrate through subcutaneous tissues toward the diaphragm.
 - (b) Larvae then find their way to the submucosa of the esophagus (*H. lineatum*) and the spinal cord or epidural fat (*H. bovis*).
 - (c) Warble stage. During January and February, larvae migrate to the dorsum of the body and reach subcutaneous tissues of the back, where they create a breathing hole and molt (L₂ and L₃). This time period may vary and is often earlier in lower latitudes. This "warble stage" lasts approximately 30 days.
 - (d) In the spring, mature larvae wriggle out of the cystic nodules and fall to the ground to pupate.
- d. Diagnostic plan. The history, physical examination findings, and the demonstration of larvae confirm the diagnosis.
- e. Therapeutic plan
 - (1) Most commonly, a topical organophosphate preparation is applied to kill migrating larvae in the early stages. This timing varies, depending on the geographical location. Later dosing runs the risk of being ineffective against L₃ larvae or causing reactions to the dead parasite in sensitive locations (spinal cord, esophagus).
 - (2) Ivermectin has also proven to be very effective.
- 2. **Screwworm** infestation
 - a. Patient profile. Screwworms are seen in all domestic animals in range or extensive management conditions in subtropical or tropical climates. Infestation occurs most commonly when fly numbers are highest in the spring, summer, and fall.
 - b. Clinical findings include extensive damage to skin, subcutaneous tissue, and muscle, accompanied by a copious amount of brown discharge and a foul odor. Animals are initially irritated by the fly strike, and then they become pyrexia, anorexia, and depressed. Death may ensue.
 - c. Etiology and pathogenesis
 - (1) Etiology. Larvae of *Cochliomyia hominivorax* and *Chrysomya bezziana* cause screwworm disease.
 - (2) Transmission. These flies lay eggs at the site of fresh wounds (e.g., from trauma, castration, dehorning) or at sites of soiling and moisture (e.g., eyes, perineum).
 - (3) Life cycle. Larvae hatch and mature in 1 week, fall to the ground, and pupate.
 - (4) Disease progression. At the site of the wound, maturing larvae feed and burrow into living tissue, producing a pronounced liquefactive, necrotic defect. Secondary bacterial infection and simultaneous fly strike with other maggots occur. Animals die from this profound tissue necrosis and secondary bacterial infection with accompanying toxemia and dehydration.
 - d. Diagnostic plan. The diagnosis is made on clinical examination; however, the condition may be well advanced before it is noticed because of coat (wool) cover.
 - e. Therapeutic plan. Infestation with screwworms is a federally reportable disease in the United States.
 - (1) Early treatment of individuals is necessary and consists of clipping and debridement of affected areas, cleansing the area, and applying insecticide preparations and antiseptic creams to the site of infestation. Many insecticides are effective and should coat the site to provide residual activity.
 - (2) Symptomatic therapy also may be necessary (e.g., antibiotics, fluids).
 - f. Prevention. The United States has mounted a very successful screwworm eradication program based on the sterilization and subsequent release of male flies to copulate with females that mate only once. (Sterilization of male flies is accomplished by using cobalt 60.) Consequently, *C. hominivorax* has been eliminated.
 - (1) Delaying surgical procedures until after fly season should be considered.
 - (2) Prophylactic wound dressings (insecticidal and antiseptic) should be used on surgical sites.
 - (3) Animals should be observed closely after surgeries or during calving or lambing seasons.
 - (4) Ivermectin should be used at the time of surgery.
- 3. Cutaneous myiasis (blowflies)
 - a. Patient profile. The disease is relatively minor in North America but causes significant animal suffering and economic losses in the major sheeprearing areas of Australia and New Zealand.
 - b. Etiology and pathogenesis
 - (1) Etiology. Flies lay eggs on animals in areas of moisture and warmth (e.g., the perineum) or decaying flesh. Flies of highest concern are *Calliphora*, *Lucilia* species, and *Phormia* species.
 - (2) Pathogenesis. Eggs hatch, and larvae feed mainly on tissue debris and decaying matter but also release proteases that allow the penetration of epidermis, leading to the extension of the wound bed and tissue damage. Secondary bacterial invasion and fluid loss occur. Larvae pupate on the ground or on carcasses or in wool.
 - c. Diagnostic plan. Diagnosis is based on observation and examination.
 - d. Therapeutic plan. Treatment includes clipping or shearing affected sites, cleaning up the wounds, and debridement of necrotic tissue. Insecticides, antibiotic creams, and lotions should be applied.
 - e. Prevention revolves around preventing the matting of haircoats with moisture and debris, delaying surgeries until after fly season, and practicing fly control.

E. Helminth infestation

- 1. Habronemiasis (**summer sores**)
 - a. Patient profile. Habronemiasis is a skin condition of horses and is seen sporadically in the spring and summer. This disease is most common in adult animals inhabiting warm climates.
 - b. Clinical findings. Skin lesions (single or multiple) are characterized by granulomatous inflammation, ulceration, intermittent hemorrhage, serosanguinous exudate, and exuberant granulation. There is mild to severe pruritis. Small (1 mm in diameter), yellowish granules are seen in diseased tissue; these granules represent necrotic calcified foci surrounding the larvae. Lesions are found on the legs, ventrum, prepuce, and in the urethral fossa and medial canthus of the eye.
 - (1) If found in the urethral process, habronemiasis may cause dysuria or polyuria.
 - (2) If found in the conjunctival sac, yellowish, gritty plaques on the palpebral and bulbar conjunctivae, eyelid granulomas, and blepharitis are present.

- (3) Gastric and pulmonary locations for *Habronema* species are asymptomatic.
 - c. Etiology and pathogenesis
 - (1) Etiology. *Habronema muscae* and *Draschia megastoma* are the adult worms of the major pathogenic larvae.
 - (2) Pathogenesis
 - (a) Adults normally inhabit the equine stomach.
 - (b) Eggs and larvae are passed in the feces and are ingested by maggots of intermediate hosts (e.g., *Musca domestica*, *Stomoxys calcitrans*). Infectious larvae are deposited on the horse, particularly in moist areas or open wounds while flies are feeding.
 - (i) Larvae that are deposited near the mouth are swallowed and complete the life cycle in the stomach.
 - (ii) Those deposited on the nose migrate to the lungs.
 - (iii) Larvae deposited in wounds or moist areas of the body produce both a local inflammatory and allergic reaction.
 - d. Diagnostic plan. The diagnosis is aided by a complete history and a physical examination.
 - e. Laboratory tests
 - (1) Deep **scrapings** or smears of lesions, particularly if yellow granules are retrieved, may reveal larvae with numerous surrounding eosinophils and mast cells. However, smears are often negative.
 - (2) Biopsy reveals a diffuse dermatitis with eosinophils and mast cells. There will be a multifocal coagulation necrosis, containing few to many nematode larvae.
 - f. Differential diagnoses include bacterial or fungal granulomas, equine sarcoid, squamous cell carcinoma, and exuberant granulation tissue. Also, *Habronema* and *Draschia* larvae will invade secondarily sarcoid, squamous cell carcinoma, and other infective granulomas, which may cause errors in diagnosing the primary skin disorder.
 - g. Therapeutic plan. Local and systemic therapy should be combined.
 - (1) **Local** therapy
 - (a) Surgery. Massive or refractory lesions should be surgically debulked. Cryotherapy should be employed using a double freeze-thaw cycle.
 - (b) **Topical** therapy includes combinations of larvicidal, antimicrobial, anti-inflammatory, penetrating, and protective agents that are applied daily under bandages. An example would be 0.03% echthiophate (phospholine iodide) with ophthalmic ointment (**maxitrol**) containing neomycin, **polymyxin**, and dexamethasone.
 - (2) Systemic therapy involves:
 - (a) Organophosphates (trichlorfon 22 mg/kg intravenously in 1 L of 5% dextrose, repeated in 2 weeks)
 - (b) Ivermectin (200 µg/kg orally)
 - (c) Glucocorticoids. Prednisone or prednisolone (1 mg/kg orally once daily) results in resolution in 7–14 days and are useful adjuncts.
 - h. Prevention. Fly control, including the prompt removal and disposal of manure and soiled bedding, is essential. Adult *Habronema* should be eliminated from the stomach by using injectable ivermectin. Any wounds should be treated symptomatically and early.
2. Onchocerciasis
 - a. Patient profile. This condition is seen most commonly in horses 4 years and older. The infestation is nonseasonal but occurs more severely in warm weather.
 - b. Clinical **findings**
 - (1) **Lesions** occur on the face and neck (particularly near the mane), the ventral chest, and the abdomen. Lesions vary from focal annular alopecia, scaling, and crusting to widespread alopecia, erythema, ulceration, oozing, crusting, and lichenification.

- (2) Seborrhea sicca may be seen in some horses. Pruritus varies from mild to severe.
 - (3) Leukoderma, premature graying, and alopecia may be permanent sequelae.
 - c. Etiology and pathogenesis
 - (1) Etiology. Onchocerca **cervicalis** infests horses throughout the world.
 - (2) Pathogenesis
 - (a) Adult worms inhabit the ligamentum nuchae. Microfilariae are numerous on the ventral midline, face, and neck.
 - (b) Hosts. **Culicoides** species, gnats, and possibly mosquitos are intermediate hosts.
 - (c) Reactions. Most horses have resident populations of **O. cervicalis**, but only certain horses develop clinical signs, suggesting that cutaneous onchocerciasis is a hypersensitivity reaction to microfilarial antigens. Dead or dying microfilariae provoke the most intense inflammatory reactions in both eyes and skin.
 - d. Diagnostic plan and laboratory tests
 - (1) A physical examination will aid in the diagnosis.
 - (2) A skin biopsy is diagnostic and will show superficial to deep perivascular to diffuse dermatitis with eosinophils. Numerous microfilariae surrounded by degranulating eosinophils are visible. Because microfilariae can be found in normal equine skin, pathogenicity must be evaluated in the light of microfilariae numbers and the inflammatory reaction present.
 - e. Differential diagnoses. The condition may appear similar to **dermatophytosis**, fly-bite dermatoses (because of the seasonal incidence), sarcoptic mange, **psoroptic** mange, and food hypersensitivity.
 - f. Therapeutic plan
 - (1) Ivermectin (200 µg/kg orally) usually produces clinical remission of the skin lesions in 2–3 weeks. Some horses require one or **two** additional treatments. Ivermectin is not an adulticide, so periodic retreatment is necessary.
 - (2) Concurrent systemic **glucocorticoids** for the first 5 days of microfilarial treatment suppress much of the inflammatory reaction (and hence clinical signs) associated with the dead and dying microfilariae.
3. **Oxyuriasis** (pin worms, thread worms)
 - a. Patient profile. Stabled horses are affected most often.
 - b. Clinical **findings** include broken hairs on the tail and a "rat tail" appearance.
 - c. Etiology and pathogenesis. **Oxyuris equi** infests the cecum and colon. Adult females leave the anus and lay eggs on the perineal skin, causing variable degrees of pruritis. This irritation causes the affected animal to rub the tail base, resulting in broken hairs and excoriation (i.e., "rat tail").
 - d. Diagnostic plan. The diagnosis is made by viewing the egg clusters on the anus or by applying clear acetate tape to the anus and looking for the eggs under a microscope. The ova of *O. equi* have an operculum (a cap located on the one end) and measure approximately 90 µm × 40 µm.
 - e. Differential diagnoses. This condition may appear similar to insect hypersensitivity, **pediculosis**, choriopic mange, food hypersensitivity, drug eruption, or self-mutilation.
 - f. Therapeutic plan. Therapy is instituted by routine deworming with benzimidazoles, ivermectin, or pyrantal pamoate.
 4. Bovine **stephanofilariasis**
 - a. Patient profile. Cattle (particularly **beef**) in the western and southwestern United States are affected. Patients range in age from 6 months to adults.
 - b. Clinical findings. Papules and nodules are found as early lesions on the ventral midline. Lesions become ulcerated and crusted, and these lesions cause alopecia and leukoderma. Mild pruritus is evident.
 - c. Etiology and pathogenesis. Clinical signs are caused by **Stephanofilaria stilesi**, a filarid worm. Adults form cyst-like structures at the base of hair follicles.

TABLE 16-2. Reported Causes of Urticaria in Domestic Animals

Causes	Examples
Dermatographism	Pressure, brushing, rubbing
Contact allergies	Saddle, soap, tack
Environmental	Cold, exercise
Venoms	Insect bites, spider bites, snake bites
Medications	Penicillin, phenylbutazone, iron dextrans
Topical treatments	Parasiticides
Foods and plants	Stinging nettle
Biologicals	Strangles vaccine, tetanus or botulinum toxoids
Systemic bacterial infections	Strangles
Dermatitis	Dermatophytosis, hypodermiasis
Inhalants	Pollens, molds, chemicals

These structures become surrounded by microfilariae. *Hematobia irritans* and other flies are intermediate hosts.

- d. Diagnostic plan. Crust scrapings reveal parasites (parts of adults and microfilariae).
- e. Therapeutic plan. Ivermectin is an effective microfilaricide and reduces the numbers of adults.

VIII. IMMUNOLOGIC SKIN DISEASES

A. Urticaria (hives, heat bumps, feed bumps)

1. Patient **profile**. Urticaria seems to be most common or most often recognized in horses.
2. Clinical findings. Urticarial reactions are acute in onset and are characterized by localized or generalized discrete (0.5–5.0 cm) edematous swellings with a flattened top (i.e., wheals). Lesions usually last only a few days, but clinical signs may persist due to the emergence of fresh wheals.
3. Etiology and pathogenesis. Urticaria may have a distinct and specific cause (e.g., penicillin allergy) or be part of a systemic event (e.g., strangles). There are a variety of triggers (Table 16-2) for the immunologic event (type I or III hypersensitivity). Triggers may be allergic or **nonallergic**.
4. Diagnostic plan and laboratory tests. The clinical findings are relatively obvious, but a detailed history and physical examination are necessary to establish the precipitating cause.
 - a. A skin biopsy interpreted by a referral institution may be helpful. Specific patterns relative to causative agents may be identifiable.
 - b. In particularly stubborn cases, serum tests for immunoglobulin E (IgE) antibody to some common allergens may prove beneficial. These tests are available commercially.
5. Therapeutic plan. Any identifiable or offending agent should be removed. Follow up with symptomatic treatment with any or a combination of the following agents:
 - a. Glucocorticoids are recommended (e.g., 0.1–0.2 mg/kg dexamethasone or 0.5–1.0 mg/kg prednisone/prednisolone).
 - b. Nonsteroidal anti-inflammatory agents (**NSAIDs**) include aspirin (5 mg/kg orally), phenylbutazone (2.2–4.4 mg/kg/orally), flunixin meglumine (1 mg/kg orally or intravenously), or diethylcarbamazine (100 mg/kg orally).

- c. Antihistamines are rarely beneficial but may be indicated if pruritis is involved. Also, hydroxyzine HCl (400 mg orally three times daily to effect, for a 500-kg horse) may be effective in horses with chronic urticaria, which is refractory to steroids. The dose may be reduced gradually over time.
 - d. Epinephrine (3–5 ml of 1:1000 solution, administered subcutaneously or intramuscularly) is indicated for life-threatening angioedematous reactions.
6. Prevention. If the etiologic agent or risk factors can be established, recommendations regarding prevention are relatively straightforward. If an allergen can be identified, subsequent hyposensitization may be attempted.

B. Equine insect hypersensitivity (sweet itch, **Queensland** itch)

1. Patient profile. This pruritic dermatitis of horses seems to worsen with age. It is seen in the warmer months, usually when animals are on pasture (paralleling fly exposure). Certain breeds may be more severely affected.
2. Clinical findings. Signs may include an initial intensely pruritic papular dermatitis of the head, ears, withers, back, croup, ventral midline, and tailhead. Lesions may extend to involve the legs, groin, axillae, intermandibular space, and ventral thorax. The intense pruritis causes excoriations, crusting, and lichenification of the papules. In severe cases, alopecia and pigment disturbances occur. "Rat tail" (disheveled tail hairs) and a "buzzed-OW" mane are common.
3. Etiology and pathogenesis. The cause of this disease is a **type-II** hypersensitivity reaction to salivary antigens from *Culicoides* species (gnat), *Simulium* species (blackfly), *Stomoxys calcitrans* (stable fly), and possibly *Haematobia irritans* (horn fly). The distribution of lesions reflects the feeding areas of the flies.
4. Diagnostic plan. A presumptive diagnosis is made subjectively based on a combination of the history, physical examination, and response to therapy.
5. Laboratory tests
 - a. A skin biopsy is very helpful because it reveals superficial or deep **perivascular** dermatitis with eosinophils. Also, focal areas of epidermal necrosis may be seen.
 - b. A complete blood cell count (**CBC**) may show a peripheral eosinophilia.
 - c. Intradermal skin testing may be employed. Aqueous preparations of whole-insect antigens can be used but are not widely available. Testing can evoke immediate or delayed reactions.
6. Differential diagnoses. Other conditions that may appear similar are **ectoparasitisms** and other **hypersensitivity** dermatoses.
7. Therapeutic plan
 - a. **Insect** control. Flies should be kept out of the stables by better manure control and the use of screens sprayed with residual insecticide. Insecticide control should be applied to affected horses. Recommended insecticides include:
 - (1) Weekly permethrin or organophosphate sprays
 - (2) Ear tags sold for cattle use (cypermethrin) attached to halter or braided into mane and tail
 - b. Systemic antipruritic agents. If insect control is not effective, any or a combination of the following should be used:
 - (1) Prednisone or prednisolone (1 mg/kg orally) is given once daily until **pruritis** is controlled, then the dosage is tapered to the lowest effective alternate-day dose.
 - (2) **Hydroxyzine** (200–400 mg/kg orally 2–3 times daily) is effective in some horses. The dose may later be tapered or discontinued after fly season.
 - (3) **Hypersensitization**. The use of commercial aqueous whole-flea antigen (flea antigen 1:1000 weight/volume) has been successful. One ml is administered intradermally until effective (3–8 weeks). Booster injections are given as needed every 1–2 months.
 - (4) Antihistamines, diethylcarbamazine, and tranquilizers have not proven effective.

C. Pemphigus foliaceus

1. Patient profile. Pemphigus has been reported in goats and horses (particularly in Appaloosa horses).
2. Clinical findings
 - a. Lesions begin on the face (especially at mucocutaneous junctions) and extremities, then become widespread. In some horses, the disease affects only the coronary bands. Transient vesicles or pustules are followed by crusting, scaling, and oozing and annular erosions bordered by epidermal colarettes. Signs of pain or pruritis are variable.
 - b. There may be concurrent signs of systemic illness (e.g., pyrexia, depression, anorexia, weight loss) in 50% of affected animals.
3. Etiology and pathogenesis. This autoimmune disease of the skin is characterized histologically by intraepidermal acantholysis and immunologically by autoantibody against the glycocalyx of keratinocytes. The result is the detachment of epidermal cells.
4. Diagnostic plan. Physical examination findings are suggestive, but the diagnosis depends on laboratory confirmation.
5. Laboratory tests
 - a. Direct smears from intact vesicles or pustules reveal numerous acanthocytes, nondegenerate neutrophils or eosinophils, and no intracellular bacteria. Occasional acanthocytes are seen in any suppurative condition, but when numerous and present in clusters, they are strongly indicative of pemphigus.
 - b. Skin biopsy of intact pustules or vesicles reveals subcorneal acantholysis with resultant cleft, vesicle, or pustule formation. Within vesicles, cells from the stratum granulosum are seen attached to overlying stratum corneum. Neutrophils or eosinophils predominate in vesicles or pustules.
 - c. Direct immunofluorescence testing reveals diffuse intercellular deposition of immunoglobulin, complement in the epidermis, or both. Glucocorticoid therapy can cause false-negative results. False-positive results can occur in equine dermatophilosis.
 - d. Indirect immunofluorescence testing for pemphigus-like antibodies is frequently positive (titers 1:10 to 1:8000). However, titers can also be demonstrated in the serum of normal horses and horses with dermatophilosis or lymphosarcoma.
6. Differential diagnoses include dermatophytosis, dermatophilosis, bacterial folliculitis, and sarcoidosis.
7. Therapeutic plan. Therapy is expensive and may have to be maintained for life.
 - a. High doses of systemic glucocorticoids is the initial treatment of choice (0.2 mg/kg dexamethasone intramuscularly as an initial treatment followed by 1 mg/kg prednisone or prednisolone orally, twice daily). Control may be obtained in 7–10 days, then alternate morning therapy should be started with the lowest dose of oral glucocorticoid possible for the elimination of clinical signs.
 - b. It may be necessary to add or substitute immunomodulating drugs if glucocorticoids prove unsatisfactory. An example is aurothioglucose (i.e., gold therapy) with the recommended regimen of:
 - (1) Two intramuscular test doses 1 week apart (5 and 10 mg for goats; 20 and 40 mg for horses), plus systemic glucocorticoids
 - (2) Followed by aurothioglucose (1 mg/kg) given weekly until there is a response (6–12 weeks)
 - (3) Followed by monthly aurothioglucose administration
 - c. Adverse effects of chrysotherapy in humans include dermatitis, stomatitis, blood dyscrasia, and proteinuria. There are no reported side effect in horses or goats; however, the hemogram should be checked biweekly and urinalysis checked weekly during induction, then every 2–3 months during maintenance.



ENDOCRINE DERMATOSES. Endocrine diseases that have cutaneous manifestations are discussed in Chapter 10.



MISCELLANEOUS DERMATOSES disorders include dermatoses for which the etiology and pathogenesis are unknown or multifactorial.

- A. Equine **eosinophilic** granuloma with collagen necrosis (nodular necrobiosis, nodular **collagenolytic** granuloma, acute collagen necrosis, eosinophilic granuloma)
 1. Patient profile. This condition is described in horses, usually during the warm months of the year.
 2. Clinical findings. Single or multiple lesions (2–10 cm in diameter) affect the neck, withers, and back. Lesions are rounded, well circumscribed, firm, nonalopecic, nonulcerative, and are not painful or pruritic. Occasionally, cystic or plaque-like lesions are seen and may discharge a central, grayish white caseous core.
 3. Etiology and pathogenesis. The cause is unknown. This disorder is likely a hypersensitivity reaction, but the insulting cause is highly speculative (e.g., antigens such as insect bites or trauma caused by saddle and girth).
 4. Diagnostic plan and laboratory tests. The history and physical examination is supported by a skin biopsy, which shows multifocal collagen degeneration with granulomatous inflammation containing eosinophils. Older lesions have marked, dystrophic mineralization.
 5. Therapeutic plan
 - a. With solitary or few lesions, surgical excision or sublesional glucocorticoids are recommended. The steroids used are triamcinolone acetonide (3–5 mg/lesion) or methylprednisolone acetate (5–10 mg/lesion).
 - b. With multiple lesions, prednisone or prednisolone (1 mg/kg orally, once a day for 2–3 weeks) is administered.
 - c. Relapses occur periodically, and older, mineralized lesions respond poorly to glucocorticoids and have to be excised.
- B. Equine **pastern dermatitis** (greasy heel, scratches, mud fever, **verruccous dermatitis**)
 1. Clinical findings. Lesions are bilaterally symmetrical and begin on the caudal aspect of the pastern, particularly of the hind limbs. Erythema and edema progress to exudation and crusting. Secondary bacterial infection and seborrhea are common complications. Pain and pruritus are variable. In chronic cases, thickening of skin, fissures, exuberant granulation tissue, severe limb edema, and lameness may be seen.
 2. Etiology and pathogenesis. Equine pastern dermatitis is not a specific disease; rather, it is a cutaneous reaction pattern of multifactorial etiology. The condition may be associated with several of the following conditions:
 - a. Staphylococcal **folliculitis**
 - b. **Dermatophilosis**
 - c. Dermatophytosis
 - d. Chorioptic mange
 - e. Primary irritant **contact dermatitis**
 - f. **Contact** hypersensitivity
 - g. Photosensitization
 - h. Pemphigus foliaceus
 3. Therapeutic plan.
 - a. Symptomatic treatments include gentle clipping and cleansing, topical application of astringent or antiseptic soaks (if the skin is already moist), or emollient ointments and creams (if the skin is dry and thickened).

- b. Definitive treatment. The specific causes, if identifiable, should be treated.
 - (1) Topical treatments include povidone-iodine rinses and metronidazole.
 - (2) Other treatments include systemic antibiotics, systemic glucocorticoids, NSAIDs, and environmental hygiene.
 - (3) Surgery. Occasionally, the condition is chronic and nonresponsive to therapy. Chronic cases with exuberant granulation tissue may require surgical excision.

XI. NEOPLASTIC DISEASES

A. Introduction

1. Patient profile. Generally, the risk of developing cutaneous neoplasia increases with age, except in horses. Specific sex and species predilections for neoplasms exist in large animals. Examples include:
 - a. Udder papillomatosis, squamous cell carcinoma in female goats
 - b. Squamous cell carcinoma in female sheep
 - c. Scrotal hemangiosarcoma in male swine
 - d. **Mastocytoma** in male horses
2. Types of cutaneous neoplasms. The most common cutaneous neoplasms (listed by the animal affected and in the approximate descending order) are:
 - a. Cattle: papilloma, squamous cell carcinoma, melanoma, mast cell tumor
 - b. Horses: sarcoid, papilloma, squamous cell carcinoma, melanoma
 - c. Swine: melanoma, hemangioma, squamous cell carcinoma
 - d. Sheep: squamous cell carcinoma, papilloma
 - e. **Goats**: squamous cell, papilloma, melanoma
3. Clinical findings vary with the type and location of the tumor.
4. Diagnostic plan and laboratory tests. In general, the diagnosis of cutaneous neoplasia is based on the history and clinical signs, **exfoliative** cytology (aspiration and impression smears), excisional biopsy, and histopathology.
5. Therapeutic plan. Clinical management may include surgery, **cryosurgery**, electro-surgery, hyperthermia, radiation therapy, chemotherapy, immunotherapy, or combinations of these treatments.

B. Epithelial neoplasms. **Papillomatosis** (fibropapillomatosis, warts) generally is seen in young animals (less than 18 months old). An exception is warts appearing on the teats of cows; these warts increase in prevalence with age. Warts are unsightly and of concern in show animals. They may also interfere with normal body functions if prominent (e.g., breeding, eating, milking).

1. Clinical finding
 - a. Cattle, sheep, and goats
 - (1) Location. Warts are found most commonly on the head, neck, and shoulders. Warts on the teats are usually multiple, small, and in various shapes. Warts may also be found in the **perineal** or genital area. There is an alimentary form that can affect the mouth, esophagus, and rumen.
 - (2) Appearance. Warts may be 1 mm to more than 1 cm in diameter and have a dry, grey, proliferative appearance. Warts may be **attached** to the skin by a wide base or with a pedunculated stem. They are uncommon in sheep.
 - b. Horses. In animals less than 3 years of age, multiple **verrucous** lesions are common on the muzzle and genitalia. Aural plaques (papillary **acanthoma**, hyperplastic dermatitis) are also found. These are bilateral lesions located on the inner surface of the ear, and, less commonly, around the vulva and anus. These lesions progress from small, smooth depigmented papules and plaques to larger, hyperkeratotic plaques.

2. Etiology and pathogenesis
 - a. Etiology. Papova viruses (PVs) are the demonstrated causes of warts in all species but goats and swine. PVs are generally host-specific. Bovine papova virus (BPV) 1, 2, and 5 can cause fibropapillomas and fibroblastic skin tumors. BPV 3–6 cause epithelial papillomas.
 - b. Pathogenesis
 - (1) Transmission. Papillomatosis is infectious and is transmitted by direct and indirect contact (i.e., animal to animal or via implements such as tattooing instruments or ear-tagging pliers). Infection requires damaged skin (from trauma, ectoparasites, UV light). In the specific case of aural plaques in horses, it is assumed that transmission is by **blackfly** bites.
 - (2) Disease progression. The virus attacks the basal cells of the epithelium and causes hyperplasia of epithelial tissue (papilloma) or proliferation of connective tissue (fibropapilloma). Fibropapillomas are the common form of papillomatosis in cattle, whereas papillomas are the common form of warts in horses.
3. **Diagnostic** plan
 - a. The presumptive diagnosis is made on the clinical picture and confirmed by histopathology if any doubt exists with the clinical picture.
 - b. **Histopathology.** If there is only epithelial proliferation, squamous papilloma (e.g., a typical equine papilloma) should be considered. With equine aural plaques, there is concurrent epidermal hypomelanosis.
4. **Therapeutic** plan. Aural plaques are persistent and, because of conformation and location, do not lend themselves to removal. For other warts that persist or in cases where a more rapid resolution is desired, consider:
 - a. Cryosurgery or surgical excision. This approach may also stimulate regression in warts that are not excised.
 - b. **Podophyllum** 50% (applied daily until remission)
 - c. **Intradermal** injections of **autogenous** or commercial wart vaccines. The response of the low, flat warts without stalks (particularly if located on the teats) is poor.
5. Prognosis. Typical papillomatosis in horses, cattle, and sheep, and papillomatosis of the head, neck, and forelegs in goats regress spontaneously in 1–6 months. Warts in other locations (e.g., glans penis or alimentary tract) carry a much less favorable prognosis. The prognosis is also poor if more than 20% of the body is affected.
6. Prevention. Although warts are difficult to prevent, some recommendations include isolating affected animals, decreasing cutaneous environmental injuries, and disinfecting the environment and equipment using formaldehyde or lye.

C. Squamous cell carcinoma (SCC)

1. Patient profile. SCC is a tumor that affects all large animals except swine. Usually seen in mature animals, this disease is of significant economic importance in cattle because of the relative frequency with which it occurs around the eye of range cattle (see Chapter 12 III B). It is also one of the more frequently diagnosed tumors of the skin of horses.
2. Clinical finding
 - a. Horses. SCCs commonly occur on the head, at mucocutaneous junctions, and on male and female genitalia. Tumors begin as solitary lesions, usually as **non-healing**, enlarging, granulating erosions (or ulcers) or as proliferative, cauliflower-like masses. Necrosis and a foul odor is common. Tumors are usually slow growing and invade rather than metastasize; up to 80% may regress. SCCs of the equine penis or prepuce are more aggressive and metastasize early.
 - b. **Cattle.** The most common SCC in cattle is the ocular form (i.e., cancer eye). Early lesions are grey-white plaques of tissue on the conjunctiva or nictitating

membrane. The majority of these lesions may regress, but many develop into papillomas of the orbit, eyelids, or periorbital skin. These are firmly and widely attached and result in irritation and lacrimation. They may become secondarily infected.

3. Etiology and pathogenesis. SCCs develop from squamous epithelium, and their genesis is related to chronic exposure of poorly pigmented, poorly haired skin to UV radiation. There may also be genetic factors (e.g., Herefords with cancer eye). Papilloma virus may be associated with the development of SCC because the virus has been found in SCC precursor lesions. Also, DNA of the papilloma virus has been identified in the tumors of cattle with SCCs.
4. Diagnostic plan and laboratory tests. Exfoliative cytology from smears of the tumor is helpful. A tumor biopsy reveals irregular masses or cords of epidermal cells that proliferated downward and invade the dermis. Other findings include keratin formation, horn pearls, intercellular bridges, and mitoses.
5. Therapeutic plan (see also Chapter 12 III B 4)
 - a. Surgical excision of the lesions, **cryosurgery**, or both is the treatment of choice. Hyperthermia (commercial portable units are available and affordable) has proven to be very useful.
 - b. Immunotherapy. Surgical procedures may be combined with immunotherapy, such as repeated intratumor injection (every 2–3 weeks) of the bacillus of Calmette-Cuerin (BCG) or a vaccine (see Chapter 12) of fresh tumor extract. Because there is a risk of anaphylaxis with repeated injections of BCG, the patient should be pretreated with **flunixin** meglumine immediately before intralesional injection.
6. Prevention. For cancer eye in cattle, a breeding program should be established to increase pigmentation around the eyes of susceptible cattle (e.g., Herefords).

D. Mesenchymal tumors (equine sarcoid)

1. Patient profile. Equine sarcoid can affect any age and breed of horse, but the majority of affected horses (70%) are younger than 4 years old. Appaloosa, Arabian, and Quarter horses are overrepresented and may have a genetic predisposition. There also may be a genetic predilection associated with the major histocompatibility complex.
2. Clinical finding. Sarcoid is the most common neoplasm of the horse. Sarcoids usually occur on the head (periocular, pinnae, commissures of lips), legs, and ventral trunk. In many horses (30%–50%), they are multiple. There are five basic gross types of sarcoids:
 - a. Verrucous (warty): may be sessile or pedunculated
 - b. Fibroblastic (proud flesh-like): sessile or pedunculated
 - c. Mixed type: **verrucous/fibroblastic**
 - d. Occult (flat): annular areas of alopecia with scaling and crusting
 - e. Subcutaneous nodules: usually located around the genitalia
3. Etiology and pathogenesis. Equine sarcoid is caused by a virus, possibly a PV (the type is yet undetermined). The lesions are moderately malignant but may remain static for years before undergoing a spurt of growth. These lesions do not metastasize but are locally invasive.
4. Diagnostic plan and laboratory tests. Histopathology is often necessary to achieve a final diagnosis and will reveal fibroblastic proliferation, epidermal hyperplasia, and dermoepidermal activity.
 - a. Collagen **fibers** and fibroblasts in the dermis are whorled, tangled, or occasionally in a herringbone pattern.
 - b. Tumor cells are spindle-shaped, fusiform to stellate, often with **hyperchromasia**, atypia, and mitoses.
 - c. Fibroblasts at the dermoepidermal junction orient perpendicularly to basement membrane in a picket fence pattern. **Overlying** epidermis is **hyperplastic** and hyperkeratotic.

5. Differential diagnoses. The clinical expression of the sarcoid may appear similar to many cutaneous masses in the horse.
 - a. Verrucous sarcoid—papilloma, SCC
 - b. Fibroblastic sarcoid—SCC, exuberant granulation tissue, habronemiasis, infectious granulomas (e.g., pythiosis, zygomycosis, bothriomycosis, mycetoma)
 - c. Occult sarcoid—dermatophytosis, dermatophilosis, demodicosis, staphylococcal folliculitis, onchocerciasis
 - d. Subcutaneous nodules—folliculitis, nodular necrobiosis
6. Therapeutic plan
 - a. **Immunotherapy** with commercial BCG vaccine (a mycobacterial cell wall preparation in oil) has been very effective for sarcoids, especially with **periocular** lesions.
 - (1) The vaccine is administered intralesionally every 2–3 weeks for approximately four treatments. Necrosis and the ulceration of lesions may occur following treatment.
 - (2) Side **effects** include an occasional malaise or anorexia. Fatal anaphylaxis has also been reported following repeated injections of commercial BCG, so pretreatment with **flunixin** meglumine and prednisolone are recommended.
 - b. Cryosurgery, radiotherapy, and **hyperthermia** are also advocated for sarcoid treatment and have met with favorable results, particularly with singular lesions. Static occult and verrucous sarcoids are best left alone because the trauma of biopsy or surgical excision often causes increased growth and aggressive behavior of the tumor.

E. Melanocytic neoplasms. Melanomas are most common in aged horses (particularly Arabians and Percherons with a gray coat color) and certain breeds of swine (e.g., Sinclair miniature, Duroc-Jersey).

1. Clinical findings. Horses present with solitary or multiple, dermoepidermal or subcutaneous masses. Firm and nodular, these masses may also be alopecic, ulcerated, and grossly hyperpigmented. They are most **often** present on the **perineal** region, undersurface of the tail, the pinnae, the periocular region, and distal limbs.
2. Pathogenesis. Melanomas may exhibit one of three growth patterns:
 - a. Slow growth for years without metastasis
 - b. Slow growth for years with sudden rapid growth and malignancy
 - c. Rapid growth and malignancy from the onset
3. Diagnostic plan. The diagnosis is confirmed by a biopsy and the histopathology of a suspect lesion.
4. Therapeutic plan
 - a. Early radical surgical excision or cimetidine (2.5 **mg/kg** orally, three times daily for 3 months), an **H₂** histamine antagonist, has been used for the clinical management of melanomas. Tumors may reduce in size by up to 50%. Treatment should be given for 2–3 weeks after there has been no further measurable decrease in the tumor size. Treatment is most helpful if used before or in conjunction with surgical management
 - b. **Intralesional cisplatin** has also been used and is usually successful only in the reduction of tumor size.

XII. HEREDITARY DERMATOSES. A partial list of hereditary conditions that affect the skin of domestic animals follows. Readers are invited to review other texts for complete description of clinical findings.

- A. Bovine porphyria is a congenital defect of porphyria metabolism, resulting in the accumulation of porphyrins in all tissues. High levels in the skin result in a photosensitivity dermatitis.

- B.** Bovine **protoporphyria** is similar to bovine porphyria but is a milder disease.
- C.** Hypotrichosis is a congenital condition of cattle, sheep, and pigs that causes partial or complete hair loss. There may be other associated defects (e.g., hypothyroidism, seborrhea, anodontia, poor growth rates).
- D.** Reticulated **leukotrichia** of horses is likely hereditary and is seen usually in yearling Quarter horses. Linear crusts on the back line result in the characteristic crosshatched patterns of alopecia.
- E.** Ichthyosis has been recorded as a hereditary and a congenital disease in certain cattle (e.g., Holstein). This disease is characterized by alopecia and a scaled skin appearance. Most animals with this disease are euthanized.
- F.** **Epitheliogenesis imperfecta** has been recorded in calves, lambs, pigs, and foals. An inherited and congenital absence of skin causes death in affected animals.
- G.** **Epidermolysis bullosa** is a congenital disease of sheep and cattle that is characterized by the development of epidermal bullae in young animals.
- H.** Baldy **calf syndrome** (inherited epidermal **dysplasia**) is an inherited, congenital defect of the Holstein breed. The disease is characterized by alopecia and the failure of horn growth. The condition is fatal because affected animals fail to grow.
- I.** **Dermatosparaxis** is an inherited disorder of cattle, horses, and **sheep**. The disease is characterized by increased skin and connective tissue fragility. The skin of affected animals is very vulnerable to trauma.
- J.** **Dermatosis vegetans** of **swine** is an inherited disorder of the skin and coronets of the feet in young pigs. Clinical signs include erythema, edema, and crustiness. Some pigs may recover, but many affected pigs die from an associated giant-cell pneumonitis.

STUDY QUESTIONS

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the ONE numbered answer or completion that is **BEST** in each case.

- Which one of the following statements regarding primary contact dermatitis is true? Primary contact dermatitis:
 - requires previous sensitization to the offending agent.
 - often develops at body locations which trap moisture.
 - is invariably pruritic.
 - is treated with corticosteroids as a first order of treatment.
 - will not resolve when removal of the offending agent.
- The cellular damage that is produced in cases of photosensitization is caused by:
 - (1) molecular excitation.
 - (2) sunburn.
 - (3) high levels of cellular chlorophyll.
 - (4) lack of skin pigmentation.
 - (5) renal disease.
- A dairy herd presents with papules, scabs, and granulomas on the teats of the cows. The condition appears to be chronic. The most likely diagnosis is:
 - horsepox.
 - bovine ulcerative mammillitis.
 - pseudocowpox.
 - contagious ecthyma.
 - bovine mucosal disease.
- Staphylococcal organisms may be implicated in which grouping of skin infections?
 - Purpura hemorrhagica, folliculitis, furunculosis, acne
 - Udder impetigo, pastern folliculitis, furunculosis, abscesses
 - Greasy heel, pigeon breast, purpura hemorrhagica, acne
 - Pigeon breast, caseous lymphadenitis, udder impetigo, lumpy jaw
 - Lumpy jaw, caseous lymphadenitis, pastern folliculitis, Canadian horsepox
- Which of the following statements regarding dermatophilosis is true? Dermatophilosis is:
 - a synonym for dermatophytosis.
 - extremely pruritic in its early active stage.
 - most prevalent in dry, arid environments.
 - diagnosed by microscopic examination of stained smears from active lesions.
 - best treated with topical fungicides.
- Which statement regarding ringworm infection in sheep is true?
 - Limbs are the **most** common site for the lesions.
 - Reinfection is common.
 - Young animals in confinement are most commonly affected.
 - Diagnosis is confirmed by use of a Wood's lamp.
 - There is little **zoonotic** potential compared to other species.
- Which one of the following statements regarding external parasitism of domestic animals is true?
 - Psoroptic mange is painful rather than pruritic.
 - Mite populations are highest in the summer.
 - Chorioptes** species are the only mange mites of pigs.
 - Melophagus ovinus** lives its entire life on the skin of sheep.
 - Tick populations are controlled with applications of organophosphates on infested animals.
- Which statement regarding habronemiasis of horses is true? Habronemiasis of horses:
 - is also known as "leeches" of horses in the tropics.
 - is characterized by yellowish granules within inflammatory lesions.
 - produces gastrointestinal signs more **commonly** than a dermatitis.
 - causes pruritis of the perineal area.
 - is transmitted by mosquitoes.

9. Which one of the following statements regarding papillomatosis in horses and cows is true?

- (1) Warts on the face of horses and teats of cows are most common in young animals.
- (2) Aural plaques should be removed.
- (3) Warts typically regress in 1–6 months.
- (4) The most common form of warts is fibropapilloma.
- (5) Commercial wart vaccines hasten a response.

DIRECTIONS: The numbered item in this section is negatively phrased, as indicated by a capitalized word such as NOT, LEAST, or EXCEPT. Select the ONE numbered answer or completion that is BEST.

11. Which one of the following procedures should NOT be employed when taking a specimen for a suspected viral dermatitis?

- (1) Samples should be taken from more than one animal or more than one location in any individual animal.
- (2) The site should be aseptically prepared with a betadine scrub and alcohol.
- (3) Tissues should be submitted for both direct electron microscopy and virus isolation.
- (4) The sample should include the periphery of the lesion.
- (5) Samples should be protected by cool storage (4°C) in transport media.

10. Which one of the following statements regarding melanomas is true? Melanomas:

- (1) are seen most commonly in aged cattle.
- (2) are the most common neoplasia in horses.
- (3) are caused by a virus.
- (4) are treated successfully with intralesional bacille Calmette-Guérin (BCG).
- (5) may grow very slowly for years.

ANSWERS AND EXPLANATIONS

1. The answer is 2 [II C 3 b]. Moisture is an important predisposing factor to the development of primary contact dermatitis. Previous sensitization to the agent is not necessary. Development of sensitivity is a function of concentrations and exposure to the agent. The dermatitis may or may not be pruritic or painful. Corticosteroids may be indicated in longstanding cases, but the first line of treatment is the elimination of the agent and the washing and rinsing of the area. Resolution usually occurs 7–10 days after the removal of the irritant.

2. The answer is 1 [II E 2 b]. Cellular damage occurs when photodynamic agents that have been deposited in the skin absorb ultraviolet (UV) light and become excited, causing cellular damage. Although a lack of skin pigmentation may increase effects of photosensitization, it is not directly causative of cellular destruction. Chlorophyll found in plants must be converted to phylloerythrin before this agent acts as a photodynamic agent. Liver disease, not renal disease, may result in photosensitization.

3. The answer is 3 [IV B 2]. This clinical description best fits pseudocowpox. As named, **horsepox** affects only horses and is a rare disease not seen outside of Europe. Bovine ulcerative mammillitis produces ulcers and teat skin sloughing. Contagious **ecthyma** is a disease of sheep and goats, affecting the mucous membranes of the nose and mouth. Bovine **mucosal** disease affects the oral cavity and the gastrointestinal tract.

4. The answer is 2 [IV A–C]. The skin conditions in which *Staphylococcus* species are implicated are udder impetigo, pastern folliculitis (greasy heel), furunculosis, acne, and many abscesses. Purpura hemorrhagica is a urticaria that is seen most commonly as secondary to *Streptococcus equi* infections. **Pigeon breast**, caseous lymphadenitis, and **Canadian horsepox** are corynebacterial infections. Lumpy jaw is an actinomycotic infection.

5. The answer is 4 [IV E 2 d, e]. Dermophilosis is a bacterial skin infection that is found most frequently under moist, humid

conditions. The condition is characterized by painful (not pruritic), crusty lesions, which leave a moist, denuded area when removed. Stained impression smears of these moist lesions or the underside of the scabs will be diagnostic for the classic appearance of the bacteria. Because this is a bacterial disease, topical fungicides have no place in the therapeutic plan.

6. The answer is 3 [VI B 2]. Dermatomycosis (ringworm) is most common in the young of any species under confinement conditions. In sheep, the head is the most likely area to be involved. Reinfection in any species is not common. Many dermatophytes do not fluoresce under a Wood's light. Sheep are no different than other species in terms of the potential to spread the condition to humans. Ringworm is a zoonosis.

7. The answer is 4 [VII C 1 c]. The sheep ked, *Melophagus ovinus*, is an obligate parasite of the host. Psoroptic mange is very pruritic and *Chorioptes* species infest a wide variety of hosts. Mite populations are usually highest at times of cool, moist conditions, usually when animals are housed in the winter months. Tick populations cannot be controlled by treatment of the host alone because these parasites live off the host for a portion of their life cycle.

8. The answer is 2 [VII E 1 b]. The ulcerative granulomatous skin lesions that are seen with the infestation by *Habronema* or *Draschia* larvae are characterized by yellow granules in diseased tissue. "Leeches" is the common name for pharyngomyiasis. Although *Habronema* and *Draschia* normally inhabit the equine stomach, they do not cause clinical symptoms. *Oxyuris equi* causes perineal pruritus, whereas *Onchocerca microfilariae* may be transmitted by mosquitos.

9. The answer is 3 [XI B 1]. Although warts are usually most common in young animals, an exception to this includes warts on the teats of cattle. It is not recommended that aural plaques be removed because of their location over the cartilaginous pinnae. In horses, the most common form of warts is

fibropapilloma, whereas papillomas are the most common form in cattle. Low, flat warts respond poorly to vaccination; therefore, vaccination is not correct in all instances.

10. The answer is 5 [XI E 21]. Melanomas are seen most commonly in aged horses. Sarcoids are seen most often in aged cattle. Sarcoids are caused by a virus and are treated with the intralesional instillation of bacille Calmette-Guerin (BCG).

11. The answer is 2 [IV A 2 c]. A diagnostician should not aseptically prepare the site before sampling it for virus isolation because disinfectants and alcohol may inactivate the virus. Samples should be taken from more than one animal or more than one location in any individual animal. The sample should include the periphery of the lesion. Samples should be protected by cool storage (4°C) in transport media. Tissue should be submitted for both direct electron microscopy and virus isolations.